THE AMERICAN ACADEMY OF ORTHOPAEDIC SURGEONS

Presents Lectures on

REGIONAL ORTHOPAEDIC SURGERY
and
FUNDAMENTAL ORTHOPAEDIC PROBLEMS

NUMBER II

Selected from the Instructional Courses of the
Fourteenth Annual Assembly, Chicago, January 25-30, 1947

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J. W EDWARDS ANN ARBOR 1948

Lithoprinted in U S A by Edwards Brothers, Inc
Ann Arbor, Michigan

FOREWORD

With the publication of this volume, the postwar era in the graduate teaching of Orthopedic Surgery enters the second phase of our realization of the need to emphasize the fundamentals of civilian practice in our specialty. Hence this volume is entitled REGIONAL ORTHOPAEDIC SURGERY AND FUNDAMENTAL ORTHOPAEDIC PROBLEMS NUMBER II. Although it duplicates some of the topics of last year's book, it amplifies them and presents the concepts and opinions of different authorities.

As these courses are evolved from year to year, it is illuminating to realize the interest expressed by the membership in having certain courses repeated. We still like to hear the old story told in different words. Perhaps therein lies the secret of the need for constant inspiration on subjects and problems with which we are familiar.

The Subcommittee for Instructional Courses (Drs. W. P. Blount, W. T. Green, C. N. Pease, C. R. Rountree, and V. P. Thompson) has solicited the membership with respect to the subjects to be covered and selected with care an outstanding faculty to give the courses. This assured the presentation of a program of general appeal.

The publication of these lectures and their widespread acceptance by the membership and those taking resident training marks an epochal step forward in the well-rounded development of graduate education in Orthopedic Surgery. I was profoundly impressed by this fact when I visited a group of hospitals this fall. I found that many of the residents relied to a remarkable extent on these published lectures as texts in the study of Orthopedic Surgery. Their regular, current publication keeps the subject material cumulative and up-to-date. As time goes on, the entire field of Orthopedic Surgery may be covered in a comprehensive and concise manner.

During the past year, in gathering and editing the manuscripts, I have been deeply indebted to Dr. Walter Blount, the Associate Editor, for his untiring and conscientious efforts. The amount of persistence and persuasion necessary to accumulate such a volume is tremendous. We hope that in the future our faculty will not only recognize the opportunity of making an outstanding contribution to graduate training by giving their courses but will realize the importance of putting them into print for future study and reference.

The editor greatly appreciates the efforts of all those who made this volume possible. I wish to thank Drs. Thomas P. and Ruth Waring and Dr. Stuart Russell of the University of Michigan Department of Orthopedic Surgery for proofreading and in particular Mr. B. A. Uhlendorf, Director of Publications, Edwards Brothers, Inc., for his splendid cooperation throughout the publication of this volume.

On behalf of the membership of the Academy and the Subcommittee for Instructional Courses, I wish to express deep gratitude to the faculty members who have made this volume possible.

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Course No. 1

BONE TUMORS

Lecturers

Henry W. Meyerding, M.D., Introduction - The Diagnosis, Treatment, & Prognosis of Primary Malignant Tumors of Bone

Dallas B. Phemister, M.D., Comments on Classification, Diagnosis, & Management of Bone Sarcomas

Mary Sherman, M.D., Osteoid-Osteoma Review of The Literature and Report of Thirty Cases

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THE DIAGNOSIS, TREATMENT, AND PROGNOSIS OF PRIMARY MALIGNANT TUMORS OF BONE

Henry W. Meyerding, M.D.

THIS IS THE second instructional course on tumors of bone to be presented under the auspices of the Committee for the Study of Bone Tumors. The first course was given in January, 1943, at which time a general review of the subject was given with the assistance of Dr. William Carpenter MacCarty, Dr. Charles P. Baker, and Dr. Herman Johnson. At that time many of you were in the service of the Armed Forces. It was the opinion of the committee that there would be considerable interest shown if some of the advances made in knowledge of tumors of bone were to be reviewed at this time. That this has proved to be true was demonstrated by the large number of applications received for this instructional course.

The committee has had scientific exhibits of moulages, roentgenograms, statistics, and motion pictures at previous meetings of this Academy. Such aids are of great value in the presentation of the instructional courses, for you must realize the difficulties which confront the speakers in attempting to cover this subject adequately in the short period allotted them. We plan to have a three-year program for 1948, 1949, and 1950 and to limit each instructional course to one special phase of tumors of bone, such as giant-cell tumors, vascular neoplasms, etc.

What Is the Tumor, Osteoid-Osteoma?

At this time special emphasis is being placed on osteoid osteoma, a term first introduced by Jaffe in 1935[1] and 1936[2] (Jaffe and Lichtenstein, 1940)[3] to designate a benign neoplastic lesion of bone which hitherto had been undescribed. Unquestionably many of these neoplastic lesions in the past have been classified as chronic sclerosing osteitis, bone abscess or cortical or spongy bone or sclerosing nonsuppurative osteomyelitis of Garré. In reporting eighty cases of chronic sclerosing osteitis in 1944, I stated "This study [Jaffe and Lichtenstein] and experience warrants careful consideration and especial pathologic investigation of material removed at operation, in regions of spongy bone frequently diagnosed as chronic osteomyelitis, or chronic sclerosing osteitis in regions of cortical bone. Fortunately the lesion is benign, the treatment is surgical excision and the results remain good irrespective of terminology employed." In the paper I adhered to the term, "chronic sclerosing osteitis," to designate a condition which I

--ve observed to affect the cortex of long nes. I have no doubt that in the future we all reclassify many of these lesions as os-oid osteoma.

Dr. Mary Sherman, who is associated with '. Phemister, will give us the results of a idy of approximately forty cases of osteoid teoma.

Dr. Dallas B. Phemister will discuss the oss and microscopic pathology of both be-gn and malignant tumors of bone and corre-te them with the clinical aspects.

Dr. Edward L. Jenkinson will review the entgenologic aspects of benign and malig-nt tumors of bone, laying special emphasis osteoid osteoma, and will discuss the value roentgen therapy.

Comments on Classification

At the present time histogenesis or the de-lopment of tissues and tumors from the un-fferentiated cells of the germ layers is em-asized in the classification of bone tumors. me physicians may wish to introduce ana-mic, histologic, pathologic, or roentgenologic riations, and this always results in a more mplicated classification. Each year in *ogress of Orthopaedic Surgery* we have pub-ished several classifications and reviewed e literature of tumors of bone. I would sug-st that those of you who are especially in-rested in this subject obtain these abstracts the current literature. In 1923 the Clinical athological Association appointed a commit-e consisting of Drs. MacCarty, Sondern, St. eorge, and Bell to formulate a classification th Drs. Bloodgood, Codman, and Ewing, and is classification was adopted by the Regis-y of Bone Sarcoma (American College of irgeons), and was modified in 1939.

The classification of tumors of bone has en and will continue to be confusing until me agreement concerning the method of assification is decided on. We need a simple, orkable classification which clearly sepa-ates the benign from the malignant groups and itlines some of the more common lesions imulating tumors of bone. In 1921, while pre-aring my paper, "Sarcoma of the Long Bones," adopted a simplified classification and pre-ented it in 1926 to the American Medical As-ociation. This classification has been modi-ed[4] and found practical and easily understood by general practitioners, pathologists, roent-genologists, and surgeons (Table 1).

Table 1

Neoplasms of Bone and Lesions Which Simulate Them

I Lesions which simulate neoplasms of bone
- A Inflammatory lesions
  - 1 Traumatic lesions (callus, ossifying hematoma)
  - 2 Infections (syphilis, tuberculosis, osteomyelitis, nonsuppurative osteomyelitis of Garré, Brodie's abcess, myositis ossificans, osteoperiostitis)[5]
- B Osteitis fibrosa cystica
- C Metabolic lesions
  - 1 Hand-Schuller-Christian disease, Gaucher's disease, Niemann-Pick disease, hyperparathyroidism
- D. Nutritional lesions
  - 1 Rickets, scurvy

II Neoplasms of bone
- A Benign osteogenic tumors
  - 1. Osteoma (exostosis)
  - 2 Chondroma
  - 3 Osteoid-osteoma
- B Fibroblastic tumors
  - 1. Benign fibroma
  - 2 Malignant periosteal and cortical tumors
- C Giant-cell tumors
  - 1 Benign giant-cell tumor
  - 2 Malignant giant-cell sarcoma
    - a Primary malignant giant-cell sarcoma
    - b Secondary malignant giant-cell sarcoma
- D Vascular neoplasms
  - 1 Benign angioma
    - a Hemangioma, cavernous or plexiform (Fibrosis - secondary)
    - b Lymphangioma (very rare)
  - 2 Malignant angioma, endothelioma
    - a Hemangio-endothelioma (Ewing's tumor, Ewing's sarcoma, multiple endothelioma, angio-endothelioma, solitary diffuse endothelioma, angio-sarcoma)
    - b Lymphangio-endothelioma
- E Malignant osteogenic sarcoma (including chondrosarcoma)
- F Multiple myeloma
- G Metastatic tumors
- H. Miscellaneous group
  - 1. Undifferentiated malignant neoplasms
  - 2 Lymphosarcoma, liposarcoma, erythroblastoma, chloroma, adamantinoma (reticulum cell sarcoma - Broders' lymphosarcoma)

Comments on Co-operation

Our knowledge of tumors of bone has and will continue to progress with the increased co-operation of the general practitioner, roentgenologist, pathologist, and surgeon. No physician in general practice can see enough patients with tumors of bone to become proficient in their care. Therefore, the general practitioner must continue to look to the consultants in larger educational centers for assistance as well as make use of the accurate information that was gained from research, such as was carried on in the past by the Registry of Bone Sarcoma. The members of the Committee for the Study of Bone Tumors and of the American Registry of Pathology offer and perform a valuable service by consulting with the medical profession and presenting programs and instructional courses such as we are offering here. Several exhibits and motion pictures have been presented. From the interest shown, it is apparent that our efforts are appreciated, we feel that it is essential that we continue such educational programs and courses.

In this age of aseptic surgery the modern facilities of the clinical, pathologic, biologic, and roentgenologic laboratories are available for all physicians. Modern communications and transportation have permitted rapid interchange of ideas and consultation in those cases in which there is any question as to the benignancy of the lesion. The roentgenograms and tissue can be sent to the physician-pathologist, who has had more experience in such cases, and thus an expert opinion may be obtained. When the time comes that a roentgenographic examination is performed of every patient who complains of a persistent painful region or swelling, we shall have made a great step forward in obtaining early diagnosis. In those cases in which a malignant lesion of bone is suspected, the routine roentgenographic examination of the thorax is imperative and will give the earliest evidence of pulmonary metastasis. With earlier diagnosis, the exclusion of metastasis, and improved methods of treatment, we may look forward to better prognosis. Too often the prognosis is hopeless because the patients come for consultation after having had inadequate treatment based on incorrect diagnosis and then the examination reveals too extensive tumefaction for removal or presence of metastasis. We must continue relentlessly in our efforts to educate the general profession to the importance of establishing early diagnosis and of carrying out adequate treatment. The Committee for the Study of Bone Tumors is ready at all times to co-operate with you in your problems of diagnosis and treatment. We welcome any suggestions as to the methods of improving the teaching value of the instructional courses.

Comments on Primary Malignant Tumors of Bone

It is obvioulsy impossible to discuss and quote from the literature material covering the opinions and statistical findings concerning all types of tumors of bone in the time allotted us here. I shall, therefore, confine my remarks to some observations on the diagnosis and treatment of the primary malignant group of tumors.[6]

Osteogenic sarcoma comprises more than half of the malignant tumors of bone which have been encountered at the Mayo Clinic. It develops from tissue that is predestined to form bone, the primitive connective tissue. Among the terms frequently used as synonyms in the past are "osteosarcoma," "chondrosarcoma," "osteochondrosarcoma," "osteochondrofibrosarcoma," "osteochondrofibromyxosarcoma," and "periosteal osteogenic sarcoma." Pathologists use these terms to describe the microscopic cell structure more accurately, and the terms may provide some indication as to treatment and prognosis. Surgeons and roentgenologists employ the useful and descriptive terms, "periosteal" and "central," or "sclerosing" and "osteolytic," to denote the location and density of the roentgenographic appearance.

Periosteal osteogenic sarcoma apparently arises from the corticoperiosteal tissue and produces true bone. Periosteal fibrosarcoma arises from the outer layer of periosteum or neighboring tissue and causes absorption or later invasion of bone but is not a primary tumor of bone. The malignant primary osteogenic sarcoma may be correctly diagnosed in most instances from the roentgenograms. I believe it is wise, however, to confirm the diagnosis by removing a specimen of the tumor, under tourniquet when possible, and having the pathologist examine the tissue and give his opinion regarding the degree of malignancy.

Roentgenograms of the thorax to exclude pulmonary metastasis should be a routine procedure before any surgical treatment is performed. The site and size of the tumor and the grade of malignancy will determine its operability. Excision or resection of the low-grade osteogenic sarcomas (grades 1 and 2) of small size may present an important advance in the treatment and prognosis. The high-grade group (grades 3 and 4), especially in the advanced cases, may permit only palliative treatment such as amputation. Not all tumors of the osteogenic type are operable at the time of treatment and frequently the patient refuses surgical treatment. In such cases roentgen rays or radium may be used in hope of a cure, but the results to date have not been encouraging.

Comments on Gradation of Degree of Malignancy

The gradation of the degree of malignancy is an important factor in determining the type of surgical treatment. We have shown that the expert pathologist by means of Broders' method[7] of grading the degree of malignancy of the lesion, after a specimen for biopsy has been obtained, can give us information of the greatest value. Broders' classification is based on the fundamental principle of differentiation of cells. The report of the pathologist may determine the fate of the extremity concerned. It may be borne in mind that osteogenic tumors may be benign or malignant and that there may be transitional forms. The benignancy or malignancy of the osteogenic tumor is not always easily determined on the basis of the clinical and roentgenologic examination alone. Before a surgeon assumes the responsibility of making a definite diagnosis, of giving his patient a poor prognosis or of subjecting his patient to loss of limb, performance of biopsy is usually advisable. The taking of a specimen for biopsy, under tourniquet, will permit microscopic study of fresh frozen or fixed frozen sections and histologic diagnosis by the pathologist. It is most important that the pathologist examine true tumorous tissue, and that he have sufficient tissue on which to base his diagnosis. I am not impressed by the value of needle biopsy, I prefer the surgical approach to the tumor whenever such an approach is possible. The co-operation of the skilled pathologist at the time of operation is of prime importance, and it is our practice to have the pathologist witness the operation, immediately examine the sections of tissue microscopically, and give us his diagnosis within a few moments, whenever possible.

In other words, we have made a great advance in the classification of tumors of bone by the method of gradation of degree of malignancy, and I believe that this consideration is of relatively more importance than is the discussion of the anatomic, roentgenologic, or genetic considerations.[8] When, after thorough study of the sections of tissue, the pathologist reports a grade 1 osteogenic sarcoma, I feel that there may be some merit in performing a more conservative type of operation, whereas, when he reports a grade 4, or high degree, malignant lesion, I would without hesitation perform amputation of the involved extremity. In some cases it is possible to excise in toto the portion of bone containing the tumor, and to insert a bone graft, without the danger of local recurrence or metastasis. We have rarely amputated an extremity in a case in which the patient had a grade 1 osteogenic sarcoma, or one of a low degree of malignancy in smaller tumors. In this latter group of cases we expect better results to follow conservative surgical treatment.[9] Again, there are the cases in which the patient has a tumor of such size that life has become unbearable; in such an instance, in the absence of visible metastasis, palliative amputation is performed for the relief of suffering. This is one of the reasons why groups of patients for whom amputation has been performed may obtain a lesser percentage of five-year cures than those patients for whom excision was the treatment.

Let us see if the foregoing statement has proved to be true. In a series of eighty-nine cases in which amputation was performed, 24.7 per cent of the patients survived for five or more years. In a series of fifty-six cases in which excision was performed, 34.1 per cent of the patients survived for five or more years Of fifty-six patients who had grade 1 and grade 2 osteogenic sarcomas, eleven patients (19.6 per cent) lived for five years or more. Of sixty-four patients who had osteogenic sarcoma of grade 3 and grade 4, eight (12.5 per cent) survived for five or more years.

Of a series of 120 patients in whom the grade of malignancy of the tumor was determined, seventeen had grade 1 osteogenic sarcoma, and

for none was amputation performed. Fifteen patients (88.2 per cent) underwent excision. On the other hand, of sixty-four patients who had tumors graded 3 and 4, thirty-nine (61 per cent) underwent amputation, and only ten underwent excision. There were thirty-nine patients whose lesions were graded 2, and seventeen (43.6 per cent) of those underwent amputation. This demonstrates that gradation of the degree of malignancy is an important factor in determining the type of surgical treatment advisable in each case of osteogenic sarcoma. In those cases in which the patient refused operation or in which the tumor was inoperable, irradiation therapy was administered, although osteogenic sarcomas[8] as a group appear to be highly resistant to radiotherapy. The value of surgical treatment may be demonstrated by the fact that of the patients treated surgically, 23.4 per cent lived five or more years, whereas, of those treated by nonsurgical methods, none survived for five years.

Fibrosarcoma

Fibrosarcoma occurred in 9 per cent of our primary malignant tumors of bone. It appears difficult and, in many instances, impossible, to state whether or not fibrosarcoma originates from bone. We included in our series of primary malignant tumors of bone all the fibrosarcomas in which primary involvement of bone was suspected on the basis of roentgenologic and, when possible, histologic examination.[11] Approximately 60 per cent of the patients were male, and about 40 per cent were female. The average age was thirty years. The femur was the location of the fibrosarcoma in 34 per cent of the cases, the tibia in 16 per cent, and 50 per cent of the lesions occurred near the knee joint. The fibrosarcomas occurring in bone appear to be more amenable to surgical treatment than are osteogenic sarcomas. Whenever possible, a specimen for biopsy is obtained, under tourniquet, fresh frozen sections are examined microscopically and the degree of malignancy is graded, after which the surgical treatment deemed advisable is performed immediately, in the majority of cases. If it were always possible to amputate the involved extremities of patients whose lesions were of a lesser degree of malignancy during the early stages of the disease, better results would be obtained. In our series of cases of fibrosarcoma, twenty-two patients underwent amputation and of this number, twenty were traced. Of the twenty traced patients, eight (40 per cent) survived five or more years. Of eight patients who underwent excision and were traced, two (25 per cent) were living five or more years later. Of eleven patients treated surgically who had lesions of grade 1 and grade 2 and who were traced, three (27.3 per cent) lived five or more years. Of seventeen patients who had lesions of grade 3 and grade 4 and who were treated surgically, fifteen were traced. Of the fifteen traced patients, four (26.7 per cent) lived five or more years. Amputation early in the growth of these fibrosarcomas, which arise from or involve bone, should offer the best prognosis for survival.[12]

Malignant Hemangio-Endothelioma (Ewing's Sarcoma, Ewing's Tumor, Diffuse Endothelioma)

Malignant hemangio-endotheliomas formerly were classified by many pathologists as small round-cell sarcomas, and histologically were often confused with inflammatory lesions, especially osteomyelitis. Increasing numbers of these lesions are being recognized by surgeons, pathologists, and roentgenologists, and have been found to be more radiosensitive than osteogenic sarcomas and fibrosarcomas. A hemangio-endothelioma usually occurs near the middle of the shaft of a long bone and spreads from the medullary cavity to absorb cancellous and cortical bone. The periosteum may form layers of new bone commonly referred to as "onionskin" formation, which is not diagnostic. Metastasis to the regional lymph nodes and lungs may occur early; later the brain and other parts of the body, as well as other bones than the primary site, may become involved. Hemangio-endotheliomas are osteolytic, and any formations of bone which seem to be in the periosteum are reactions to the stimulation of tumor growth. Vague symptoms, with pain of a fleeting character that usually is more severe at night, may give rise to the erroneous diagnosis of rheumatism. The lesion is characterized by remissions, chills, and fever; leukocytosis may occur. I have observed patients who had lesions the true nature of which was not recognized until anemia, cachexia, and metastasis had revealed the deadly character of the tumor.

In our series of malignant hemangio-endotheliomas practically 72 per cent of the pa-

tients were male, 67 per cent were less than thirty years old. The lesions occurred in an extremity in 62 per cent of the cases of our series; twenty-eight lesions (24.6 per cent) occurred near the knee joint. The bone most frequently involved was the femur, since thirty (26.3 per cent) of our series of 114 patients had a lesion in this location. Since malignant hemangio-endothelioma was not commonly recognized until 1922, and since we did not practice systematic radiotherapy for this group of primary malignant tumors of bone prior to 1920, patients who had such a lesion were treated principally by surgical methods. However, we find that increasing reliance is being placed on radiotherapy and radiotherapeutic tests. Again we emphasize the fact that microscopic verification of the clinical and roentgenologic diganosis is a most important factor in this group of tumors, and we also stress the fact that there is a favorable reaction to irradiation therapy, even though it may be temporary in some cases. As our experience increases, the importance of irradiation as a diagnostic and therapeutic agent is evident. Practically 88 per cent of the patients in our series have had the benefits of microscopic verification of the clinical and roentgenologic diagnosis of malignant hemangio-endothelioma.

In an analysis of our series of cases in this group, thirty-three patients who underwent amputation were traced. Of this number, eight (24.2 per cent) were living five or more years later. Of twelve patients who underwent excision and were traced, two (16.7 per cent) were living five or more years later. The trend toward irradiation therapy is demonstrated by the fact that forty-seven patients were treated by this method and were traced, eleven (23.4 per cent) lived five or more years. Of patients who had grade 1 and grade 2 hemangio-endotheliomas, sixteen were traced, and of this number three (18.8 per cent) were living five or more years after surgical treatment, whereas, of fifty-two patients who had lesions of grade 3 and grade 4 and were traced, eight (15.4 per cent) were living five or more years later. Seven patients who received no treatment were traced, all were found to have died within five years.

Biopsy and surgical operation are necessary and justifiable, even though there may be extensive involvement, destruction of joints, pathologic fracture, or previous operations or infections that have obscured the nature of the lesion. Early tumors may disappear after irradiation, and the bone may assume a normal appearance. The surgeon must remember that clinically, roentgenologically, and sometimes surgically, this lesion may simulate an inflammatory lesion and as a result the diagnosis may be "osteomyelitis."

Multiple Myelomas

Multiple myelomas are classified among the primary malignant tumors of bone, but we are inclined to look on these lesions more as manifestations of disease of the lymphoid system than as manifestations of bone tumor. Plasma-cell myeloma is found in the tonsils, the lymph nodes, the larynx, and the nasopharynx.[13] Although the clinical picture of multiple myelomas is vague and the early symptoms are pain, fever, and anemia, later weakness, abdominal pain, backache with osteoporosis and loss of weight develop and suggest a malignant lesion. There may be deformity of the spinal column, referred pain and pathologic fracture. Large quantities of protein have been found in the urine in 53 per cent of cases, hence, we feel that routine examination of the urine for Bence-Jones protein should be made. Symptoms may be intermittent, and periods of recession may occur. The spinal columns of elderly patients who have unexplained abdominal pain should be carefully examined. Soft tender masses may be present in the sternum, ribs, and clavicles.[14]

Multiple myelomas constitute a fatal disease, and treatment is of little avail. Surgical treatment is practically limited to the performance of sternal puncture to obtain material for blood smears, and excision of specimens for microscopic study. Irradiation has proved to be of little value in the treatment of this condition.

Malignant Giant-Cell Sarcoma

Primary malignant giant-cell sarcoma is an extremely rare tumor. We have classified it under the term "primary malignant giant-cell sarcoma" in those cases in which the diagnosis is made at the time of the first microscopic examination and in which no previous surgical or irradiative treatment has been carried out. Tumors of this group do not include those benign foreign-body giant-cell tumors originally recognized as benign lesions which sub-

sequently underwent malignant transformation. We recognize the rare osteogenic type of sarcoma which may contain numerous foreign-body giant cells that mask its true malignant nature. These primary malignant giant-cell sarcomas cannot be diagnosed on the basis of clinical and roentgenologic observations alone. The close co-operation of the pathologist and the surgeon at the time of operation, including the microscopic study of sections of fresh frozen tissue, frequently permits the diagnosis of malignancy to be made and the performance of the surgical procedure deemed advisable in one stage. There are instances in which the pathologist cannot definitely give his opinion as to the benignancy or malignancy of the lesion, and in such instances, study of sections of fixed frozen tissue is the procedure we prefer. Our experience with these tumors is very limited, and it is too early to give statistics of any great value.[15] However, the degree of malignancy and the location and size of the tumor are important factors in determining the surgical procedure that is to be employed in each case. Of seven patients we have observed with this type of lesion, six had a lesion located in both the epiphysis and the diaphysis of the lower part of the femur, one patient had a lesion located in the upper epiphysis and diaphysis of the tibia, all lesions, therefore, occurred at the knee joint. Malignant giant-cells were not observed in any of these cases. The malignant process occurred in the stroma or tissue between the foreign-body giant-cells. Three of the seven patients were dead at the time of this report, one of these three had had the benefits of biopsy and irradiation treatment, and two had undergone amputation and had received irradiation treatment, none of the three survived five years. Four of the seven were living four, ten, twelve and fifteen years, respectively, after treatment. All four had undergone amputation and had received irradiation treatment. It is my opinion that amputation, after early diagnosis and pathologic proof of malignancy, is the treatment of choice.

Summary

Surgical treatment offers the best method of treatment of primary malignant tumors of bone. Surgical treatment permits biopsy and gross and microscopic study of tissue, which leads to a more nearly accurate diagnosis. The type of surgical treatment to be employed depends on the location, the extent of the growth of the lesions, and the grade of malignancy, all of which are important factors to be taken into consideration in each case. Routine roentgenographic examination of the thorax should be performed to exclude recognizable metastasis to the lungs. Although efficient laboratory facilities which permit early diagnosis are now available, it will not be possible to give the patient who has a primary malignant tumor of bone an improved prognosis until all general practitioners carry out routine roentgenographic examination of all painful regions.

REFERENCES

1. Jaffe, H L "Osteoid-osteoma", Benign Osteoblastic Tumor Composed of Osteoid and Atypical Bone Arch Surg. 31 709-728 (Nov) 1935
2 Jaffe, H L Osteoid Osteoma Am. J Path 12 796 (Sept) 1936
3. Jaffe, H L , and Lichtenstein, L. Osteoid-Osteoma Further Experience With this Benign Tumor of Bone, with Special Reference to Cases Showing the Lesion in Relation to Shaft Cortices and Commonly Misclassified as Instances of Sclerosing Non-Suppurative Osteomyelitis or Cortical-Bone Abscess J. Bone & Joint Surg. n s. 22·645-682 (July) 1940.
4. Meyerding, H W. Classification of Bone Tumors Proc. Staff Meet , Mayo Clin 18 17-18 (Jan. 27) 1943.
5. Meyerding, H. W Chronic Sclerosing Osteitis Tr. West. S. A. 52 545-564, 1944.
6 Meyerding, H. W The Preoperative Differential Diagnosis of Bone Tumors J. A. M. A 88 365-371 (Feb 5) 1927.
7 Broders, A C , The Grading of Carcinoma. Minnesota Med 8 726-730 (Dec) 1925.
8. Meyerding H. W,, and Valls, J. E.· Primary Malignant Tumors of Bone. J. A. M. A. 117 237-243 (July 26) 1941.
9. Phemister, D. B. Rapid Repair of Defect of Femur by Massive Bone Grafts after Resection for Tumors Surg., Gynec. & Obst 80 120-127 (Feb.) 1945.
10. Meyerding, H. W , The Results of Treatment of Osteogenic Sarcoma. J. Bone & Joint Surg. n s. 20 933-948 (Oct) 1938.

11 Meyerding, H W., Broders, A. C , and Hargrave, R L. Clinical Aspects of Fibrosarcoma of the Soft Tissues of the Extremities. Surg., Gynec & Obst. 62 1010-1019 (June) 1936

12 Broders, A. C , Hargrave, Robert, and Meyerding, H. W. Pathological Features of Soft Tissue Fibrosarcoma, with Special Reference to the Grading of Its Malignancy Surg , Gynec & Obst 69 267-280 (Sept) 1939.

13 Figi, F A., Broders, A C., and Havens, F. Z Plasma Cell Tumors of Upper Part of Respiratory Tract Ann Otol , Rhin & Laryng 54 283-297 (June) 1945

14. Meyerding, H W. Multiple Myeloma Radiology 5 132-146 (Aug) 1925

15. Meyerding, H. W. and Broders, A C Primary Malignant Giant-Cell Sarcoma of Long Bones Tr West S A (1941) 51 76-95, 1942

COMMENTS ON CLASSIFICATION, DIAGNOSIS, AND MANAGEMENT OF BONE SARCOMAS

Dallas B. Phemister, M.D.

Primary sarcomas of bone vary greatly in structure, and no classification yet proposed has met with general acceptance. The stem cells of a bone may potentially give rise to malignant growths representative of each type of fixed cell or tissue that enters into its structure, excepting nervous tissue, which comes from without. Consequently classifications, such as that of the Registry of Bone Sarcoma of the American College of Surgeons reported by Ewing, are usually based on a combination of cell and tissue types.

An analysis of the Registry cases in 1939 revealed 1,437 primary bone cancers classified as follows osteogenic sarcoma 895, or 62.3 per cent (6 parosteal), chondrosarcoma 110, or 7.6 per cent, periosteal fibrosarcoma 29, or 2 per cent, Ewing's sarcoma 238, or 16.5 per cent, myeloma 112, or 7.7 per cent, reticulum cell sarcoma 10, or 0.7 per cent, lymphosarcoma 19, or 1.3 per cent, malignant giant-cell tumor 19, or 1.3 per cent, and liposarcoma 5, or 0.3 per cent. This gives osteogenic sarcoma too high a percentage because of the inclusion of certain other tumors, such as central fibrosarcoma, in that class.

By the designation of each variety according to the principal tissue or cell of which it is composed, the great majority of primary sarcomas of the bone may be fitted into the following classification, leaving a relatively small number of undifferentiated tumors unclassified (1) osteogenic sarcoma, (2) chondrosarcoma, (3) fibrosarcoma, (4) malignant giant-cell tumor or giant-cell sarcoma, (5) Ewing's sarcoma (so-called endothelial myeloma), (6) reticulum cell sarcoma, (7) lymphosarcoma, (8) angiosarcoma, (9) liposarcoma, and (10) myeloma.

Osteogenic Sarcoma

Osteogenic sarcoma is a malignant tumor whose cells possess the property of producing bone. It is the most important of the bone sarcomas, since it comprises about one half of all cases and has a high mortality rate.

The tumor nearly always starts within the bone, replacing the medullary tissue and invading and eroding the cancellous bone. The cells form tumor bone which varies greatly in amount and in degree of differentiation. In some cases the bone is scanty, in others, abundant and dense, in some cases it is little differentiated, in others it consists of well-formed trabeculae. As growth continues, the cortex is eroded and penetrated, and the sarcoma forms a mass on the surface of the bone. The periosteum may lay down new bone on the shaft beyond the limits of the tumor. In some cases the tumor outside of the bones ossifies to about the same extent and in the same manner as that within the bone. However, in other cases it ossifies much less extensively, and the new tumor bone radiates outward from the cortex into the unossified portion. Cartilage sometimes forms in the process of ossification of peripheral osteogenic sarcoma just as in the process of ossification of peripheral callus. Osteogenic sarcomas cast characteristic shadows of bony density in roetngenograms.

The external tumor may grow to great dimensions, and regional veins may be invaded even while the tumor is small. Metastases usually develop relatively early, but they may not grow to a size by which they can be recognized for one or more years. The tumor spreads not only by way of the blood stream to the lungs, bones, and other tissues but also not infrequently by way of the lymphatics to the regional lymph nodes. The metastases usually ossify if the primary tumor is decidedly ossified.

Chondrosarcoma

Chondrosarcoma of bone consists, in the greater part or all of its extent, of cartilage showing varying signs of differentiation and other signs of malignant growth. Clinically it simulates osteogenic sarcomas in many respects but usually runs a more chronic course and frequently casts characteristic shadows in roentgenograms. A soft cartilaginous tumor begins centrally and erodes cancellous bone and inner portions of cortex. There may be little or no surrounding formation of new bone, so that roentgenologically the lesion resembles other osteolytic tumors, bone cysts, and granulomas. However, if the thicker portion of the shaft becomes involved, both periosteal and endosteal new bone are laid down to form a thick, expanded shell about the central soft tumor. Roentgenologically this casts a thick, dense shadow surrounding an oblong central radiolucent area which is highly suggestive of a chondrosarcoma.

Other cases of central tumor may reach large size, and blotchy islands of calcification and ossification may appear within the cartilage. Roentgenologically they cast characteristic blotchy, dense shadows. Even more characteristic are the disconnected, irregular, dense shadows seen in roentgenograms of peripherally situated chondrosarcomas which contain islands of calcification and ossification.

Invasion of the regional veins is not uncommon in the advanced stages of chondrosarcoma, and cartilaginous pulmonary and lymph node metastases may develop.

Fibrosarcoma

Fibrous tissue is a constituent of the marrow as well as of the periosteum; fibrosarcoma of bone may begin in either structure. Contrary to previously held views fibrosarcoma is much more frequently primary in the medullary region than in the periosteum. In the extremities it usually begins in the cancellous portion of the end of the shaft and gradually destroys and replaces both medullary and cortical structures. The tumor tissue never forms tumor bone. After perforation of the cortex an oval mass forms along one side of the bone, and the adjacent periosteum may lay down small amounts of new bone. Pathologic fracture is not uncommon at this stage.

Microscopically the tumor usually consists of spindle cells and a more limited number of round cells with collagen fibers in the matrix and nuclear evidences of malignant growth. Necrosis is common. Roentgenograms usually reveal extensive reduction in density in the central region and irregularly reduced density of portions of the cortex. There is no shadow of new bone within the tumor, but there may be shadows of periosteal new bone along the surface of the adjacent shaft.

Spindle-cell fibrosarcoma of varying grades of malignancy may arise in the periosteum and produce an oval swelling with little tendency to invade the underlying bone. Ewing emphasized the remarkable tendency for some of these tumors to produce multiple metastases in the periosteum of other bones. Primary periosteal fibrosarcoma has been rare in the author's experience.

Fibrosarcoma usually pursues a clinical course somewhat less malignant than that of osteogenic sarcoma. But in most cases metastases, often not detectable clinically, are present before treatment is initiated, and the disease ends fatally months or years later.

Malignant Giant-Cell Tumor or Giant-Cell Sarcoma

Benign giant-cell tumor of bone is relativetively common, whereas malignant giant-cell tumor is rare. The Registry in 1939 contained 238 benign giant-cell tumors as against the 19 cases that were malignant. Both benign and malignant forms occur most frequently between the ages of 15 and 40 years, and in the limb bones they involve both the end of the shaft and the epiphysis.

Most giant-cell tumors that terminate as cancers have the appearance of being initially benign. They produce chronic pain in the region of the joint and later a swelling on one

side of the bone. Roentgenograms reveal a sharply circumscribed central area of reduced density beginning in the metaphysis and extending into the epiphysis, and there is no shadow of reactionary new bone. Under the diagnosis of a benign tumor the lesion is removed with the curet and often the cavity is filled with bone grafts. Pathologic examination of excised tissue usually confirms the diagnosis of a benign lesion, but in the course of months or years there is recurrence of pain and tumor, and roentgenograms exhibit more ragged destruction of bone. At a subsequent operation the diagnosis of cancer is established.

In a minority of cases the initial tumor grows more rapidly and produces an external swelling with irregular osseous destruction or a triangular layer of new bone in the neighboring periosteum, suggestive of cancer. Pathologic examination after biopsy or curettage establishes the diagnosis of cancer.

Grossly, a benign lesion is usually chocolate colored while a malignant one is grayish and mottled. The two types of tissue may be seen in the same specimen when cancer has appeared as a secondary change in a benign tumor. Giant-cell sarcoma is nearly always non-ossifying. Microscopically it contains giant-cells, most of which are of small size, and occasionally a hyperchromatic or dividing nucleus is seen. The mononuclear cells are greatly in excess and in most of the extent of the lesion, and some show hyperchromatic nuclei or various stages of cell division.

Ewing's Sarcoma (Round-Cell Sarcoma of Uncertain Origin)

Ewing separated from the poorly differentiated malignant growths of bone a tumor which he thought consisted of angioendothelial cells and which he designated as endothelial myeloma. The tumor is relatively frequent in childhood and early adult life, affects the bones of the trunk almost as frequently as those of the extremities and is predisposed to produce widespread metastases in bone. It begins in the medullary cavity, grows at varying rates and destroys bone, but its cells never produce bone. On nearing the surface it usually stimulates the periosteum to form considerable bone, but the amount may be small, especially in adults. Pathologic fracture is not uncommon in neglected cases. Widespread metastases in bone may destroy so much marrow that severe anemia is produced. Roentgenograms reveal reduction in density from wormeaten destruction of cancellous and cortical bone and a surface layer of increased density from the periosteal new bone. Metastases produce conditions similar to those of the primary tumor.

The tumor responds well to initial roentgen treatment, but there is recurrence in a high percentage of cases and this, with the frequent metastases, produces a fatal termination.

Ewing described the tumor as consisting of small polyhedral cells with pale cytoplasm, a small hyperchromatic nucleus and a definite cell border. In some cases tumor cells were thought to line vascular spaces, but other observers have regarded this appearance owing to survival of perivascular cells in degenerating portions of the tumor. Well-preserved cells have an ill-defined border, scanty cytoplasm, a relatively large nucleus, and scattered chromatin.

Much confusion exists concerning this and kindred tumors of bone marrow, and many authors deny the endothelial origin of any of them. In the opinion of Oberling, Ewing's tumor comes from the supporting mesenchymal cells of the bone marrow and is a reticulo-sarcoma. According to Stout it is a derivative of the mesenchymal supporting framework of the bone marrow and a variant of reticulum-cell sarcoma. By Lichtenstein and Jaffe it was regarded as a sarcoma derived from primitive marrow-connective tissue but without further specification. Willis pointed out that osseous metastases from neuroblastoma arising in the adrenal medulla or in sympathetic nerve ganglions located elsewhere have often been wrongly diagnosed as Ewing's sarcoma. This accounts in part for the exceedingly high mortality usually given. Further study is necessary before the slightly differentiated malignant growths arising in the medullary cavity can be classified properly.

Reticulum-Cell Sarcoma

Primary reticulum-cell sarcoma is a non-ossifying, bone-destroying tumor derived from embryonal reticular tissue of the marrow and is characterized by an unusually favorable response to surgical treatment.

It affects both long and flat bones and grows to a large size, first within the medullary region and later outside the shaft. The tumor is

gray to red, erodes the bone, stimulates the periosteum to form new bone, and becomes necrotic in its deeper regions. Grossly and microscopically it bears some resemblance to Ewing's tumor, with which it is often confused.

The tumor consists of round, oval, and elongated cells, which are somewhat larger than lymphocytes. The nucleus is relatively large and oval, and nucleoli are sometimes prominent. The stroma contains both collagen and reticulum fibers, and cytoplasmic processes are present.

Metastasis, both of weight, and cachexia appear relatively late, and amputation or wide local resection in case of trunk bones gives strikingly good results. Of Parker and Jackson's seventeen patients treated principally in this way seven were free from signs of the disease ten or more years afterward.

Lymphosarcoma

In some cases of lymphosarcoma the bones have been the seat of the first lesion to be detected. The tumor is painful and produces central destruction with little or no accompanying new bone. Microscopically the cells consist of malignant lymphocytes in a stroma containing both collagen and reticulum fibers. This has led to an endeavor to establish primary lymphosarcoma of bone as a separate disease. Wieland discussed a series of such cases as possible myelomas but preferred to regard them as reticulum-cell lymphosarcoma. Craver and Copeland reported several patients with multiple lymphosarcomatous bony lesions who later had extension of the process to the lymph nodes, thereby completing the clinical and pathologic picture of a reticulum-cell lymphosarcoma. In view of the regular appearance of lesions in both the bone marrow and the lymph nodes and the frequent multiple osseous foci, it appears advisable to classify all lymphosarcoma as a disease of the lymphatic system and not to recognize a variety of primary lymphosarcoma of bone which gives rise to metastases in the lymph nodes.

Angiosarcoma

If Ewing's tumor is excluded as being of non-endothelial nature, angiosarcoma (telangiectatic sarcoma or malignant bone aneurysm) is left as the only sarcoma of the bone which is of vascular origin. It is a rare tumor which is most frequent in childhood and adolescence, it begins in the medullary cavity, grows rapidly, erodes the bone (which is replaced by large blood sinuses), and elevates the periosteum, often creating thereby a blood-filled sac lined by tumor.

The peripheral portion consists of reddish tissue which microscopically is composed of hyperchromatic spindle and polyhedral cells, with numerous small blood sinuses scattered throughout and lined by tumor cells. The tumor reaches a large size, frequently causes fracture, metastasizes to the lungs and produces death in the course of several months.

Liposarcoma

Only a few cases of liposarcoma have been described as arising from the fat tissue of the bone marrow. Stewart and Rebboch and Hauser have reported bone-destroying tumors with malignant characteristics which give rise to metastases in other bones and in the lungs. Microscopically the structure resembles liposarcoma in other locations. Additional cases have been reported recently by Khanolkar and Williford and Fatherree.

Myeloma

Myeloma is a malignant tumor of hemopoietic elements of the bone marrow, usually multifocal in origin. It is seen principally in adult life and most frequently beyond the age of 30. Men are about twice as frequently affected as are women. The lesions first to cause pain are most often located in vertebrae, ribs, pelvis, femur, and humerus. They produce small to large punched areas of osseous destruction which gradually become confluent. Little or no new bone is formed. There is loss of weight, anemia, and generally debility as the disease advances, fractures may occur, leading to deformities.

The tissue of the tumor is soft and grayish to brown according to vascularity. The main varieties in order of frequency are plasma-cell myeloma, myelocytoma, lymphocytoma, and erythroblastoma. Bence-Jones albumosuria and hyperproteinemia are present in the majority of the patients. Roentgenograms which exhibit the multiple punched areas of reduced density are of diagnostic importance. Sternal puncture usually yields myeloma cells, but a biopsy may be necessary in order to confirm the diagnosis.

Solitary myeloma usually becomes multiple,

but it may persist as such for long periods or be controlled indefinitely by roentgen therapy without the appearance of other lesions. Roentgen therapy should also be used in multiple myeloma, as it relieves pain and prolongs life.

Diagnosis

The diagnosis of sarcoma of the bone is generally based on a history of chronic recurrent pain and, sooner or later, the appearance of a deep-seated bony swelling. Since in the extremities it is usually near a joint, the condition is most frequently mistaken for rheumatism. Roentgenograms taken during this stage reveal changes which are either highly suggestive or diagnostic of sarcoma. However, before either surgical or radiation therapy is started the diagnosis should nearly always be confirmed by biopsy.

Osteogenic sarcoma casts amorphous shadows of increased density in the medullary region, in which it has invaded and replaced old bone. More or less radiating shadows of bony density are cast along the surface of the shaft if the sarcoma has broken through the bone.

Chondrosarcoma may frequently be recognized by the presence of blotchy shadows of increased density or a thick cortical shadow of increased density surrounding a central zone of reduced density.

Medullary fibrosarcoma produces an irregularly outlined central shadow of reduced density and, if it has broken through the cortex, a peripheral shadow of faint density. There is relatively little periosteal new bone casting dense shadows along the surface.

Malignant giant-cell tumor usually progresses more rapidly and produces more pain than the benign form, and in roentgenograms the bony margin may show ragged reduction in density resembling fibrosarcoma and osteolytic metastatic carcinoma.

Ewing's sarcoma, with its irregular reduction in density of old bone and varying amounts of increased density peripherally from periosteal new bone, may simulate chronic inflammation and metastatic tumors as well as other forms of sarcoma of the bone. Onset in early life and early metastases suggest Ewing's sarcoma. Lamellated periosteal shadows are sometimes present but not characteristic.

Reticulum-cell sarcoma produces local changes similar to Ewing's sarcoma but occurs in a somewhat older age group, runs a slower course, and is much more favorably controlled by surgical procedures.

Angiosarcoma infiltrates and destroys bone, producing roentgen ray shadows of reduced density. It metastasizes early and runs a rapidly fatal course, but a biopsy is necessary to make the diagnosis.

Multiple myeloma is usually first suspected from multiple punched-out areas of reduced density observed in roentgenograms which were made because of pains in the bones in the absence of tumor. They simulate the picture of osteolytic metastases produced by carcinoma, especially from the breast. The diagnosis is usually confirmed by the observation of Bence-Jones albumosuria and hyperproteinemia, which substances are seldom present in solitary myeloma.

In the final analysis there are some cases in which the diagnosis remains uncertain and others in which one must be contented with the diagnosis of cancer without knowledge of the exact variety.

Treatment

The treatment of a sarcoma of the bone, in the absence of demonstrable metastases, should be by wide resection or excision of the involved bone, if it is located in the trunk or head and lends itself to such an operation. If located in an extremity, the treatment should usually be by amputation at a level well above the upper limits of the tumor. If the sarcoma of a long bone is located in the lower one third, amputation should be either through or above the upper one third, and if in the upper one third, amputation should be above the proximal joint or sometimes through it, as in the case of the hip. An additional safeguard, though not extensively practiced at present, is excision of the regional axillary or inguinal lymph nodes. Another more frequently used safeguard is supplementary radiation therapy, both local and regional.

The average mortality for all varieties of sarcoma of the bone that have been treated along these lines is high. Metastases frequently develop and local recurrence is not rare. Long term statistics of a large series of well-studied cases are sadly lacking, but less complete information indicates that the five-year survivals are under 15 per cent and that a fair percentage of these are permanently cured. The mortality for angiosarcoma, osteogenic

sarcoma and Ewing's sarcoma is notably high, while that of chondrosarcoma and reticulum sarcoma is definitely less so.

Irradiation is the treatment of choice in case of sarcoma of the bone which is so located that surgical treatment cannot be employed or which has produced demonstrable distant metastases. Ewing's sarcoma, lymphosarcoma, reticulum-cell sarcoma and metastatic cancer usually respond favorably to treatment but sooner or later recur and become radioresistant. Embryonal chondrosarcoma also responds favorably and may be held in check for prolonged periods. Osteogenic sarcoma responds extremely poorly, and it is rare to see a case in which prolonged palliation is obtained.

In long bones a selected group of sarcomas of the lower grades and relatively small size may be treated by extensive resection of the involved bone and adjacent soft tissues and repair of the skeletal defect by massive transplantation of bone. Experience has shown that a useful extremity may thereby be preserved. The incidence of local recurrence is only slightly to moderately increased over that which follows amputation, and the incidence of distant metastases is only increased to the extent that they may spread from a possible local recurrence. From my limited experience the most favorable types for such treatment are chondrosarcoma, giant-cell sarcoma and reticulum-cell sarcoma. (One patient remains well seven and one-third years after resection of 17 cm. of the shaft of the femur, with subsequent reconstruction by transplantations of bone.) Blood transfusion, tourniquet hemostasis, and antibiotics have rendered these as well as other extensive operative procedures more feasible. One of the most important future advances in the surgical treatment of sarcoma of the bone should come from their judicious employment and expansion.

OSTEOID-OSTEOMA REVIEW OF THE LITERATURE AND REPORT OF THIRTY CASES

Mary Sherman, M.D.

Although instances of osteoid-osteoma had been reported under other names before Jaffe's original paper, the lesion had not previously been generally recognized. Since that time many cases have been reported and the clinical features of osteoid-osteoma are by now well known.

The disorder is one which is more than twice as common in the male than it is in the female.

It has been said by Jaffe that the lesion has a predilection for adolescents and young adults. Indeed, over half the patients already reported are between 10 and 20 years old. In the present series, as is seen in Table 1, the age incidence is slightly different, with over half of the patients between the ages of 5 and 15 years.

The youngest patient in our series was three years old and the oldest 51.

In Table 2 the location of the previously reported lesions and those of the present series are compared in Table II on the following page.

Table I

Age Incidence

Age	Reported Cases	Present Series	Total
1 to 5 years	8	1	9
6 -- 10	9	9	18
11 -- 15	30	8	38
16 -- 20	35	5	40
21 -- 25	24	2	26
26 -- 30	14	2	16
31 -- 35	3	2	5
Over 36	2	1	3
Not stated	3		3
	128	30	158

It will be seen that in general the location of the lesions in the present series corresponds well with those in the literature. The only noteworthy exception is that almost 25 per cent of our series were lesions of the spine, which were more numerous than any other

Table II

Location

Bone	Cases in Literature	Present Series	Total
Tibia	33	4	37
Femur	24	7	31
Vertebrae	11	7	18
Astragalus	12	1	13
Humerus	9	1	10
Toes	6	3	9
Fingers	5	4	9
Fibula	7	0	7
Os calcis	4	0	4
Radius	3	1	4
Ulna	3	0	3
Carpal scaphoid	3	0	3
Ilium	2	1	3
Patella	1	0	1
Pubis	0	1	1
Tarsal scaphoid	1	0	1
Hamate	1	0	1
Capitate	1	0	1
Rib	1	0	1
"Ankle"	1	0	1
	128	30	158

category, probably because of our special interest in this particular disorder. In any given bone the lesion may be entirely in cancellous bone, beneath or in the cortex, or even subperiosteal.

All the patients in this series sought medical advice because of pain. Their stories are remarkably similar The pain, which at first was rather vague, mild, and intermittent, gradually grew in severity and constancy and localized to a very small area. Twenty-four of the thirty patients volunteered that the pain was most severe at night and often prevented sleep or awakened them. Three patients had had symptoms for only two months, twenty-one from six months to two years, and the remaining six for more than two years. One patient had had unremitting pain for six years. When the lesion is in the spine, there may be associated root pain and even symptoms of cord compression.

The chief physical finding is that of tenderness which is usually sharply localized, even by the very young patients, and may be exquisite. If the lesion has been present long enough, there is usually palpable thickening of the bone. Lesions in bones with little soft-tissue covering may be accompanied by swelling, but this is rarely warm and never red. If the site is near a joint there is often limited and painful motion of that joint. Occasionally there is fluid in the joint and the findings may be such as to simulate a primary arthritis. Involvement of the lower extremity usually produces a limp and atrophy of the muscles. In the spine osteoid-osteomas produce all the signs of acute localized back pain, including muscle spasm, secondary scoliosis, and pelvic tilt.

There are no systemic complaints or findings. The patients are afebrile and have a normal blood count.

Roentgenographic examination of a mature lesion reveals a characteristic picture. The active nidus is usually a small round or oval area of reduced density. Often one can see within it a small dense shadow which represents ossification of the central portion. About this nidus is almost always a thick dense shadow of sclerotic regional bone. If the lesion is at or in the cortex, the regional hypertrophy, especially along the periosteal surface, is much greater. The circumference of the shaft may be so greatly increased and so sclerotic that the nidus is difficult to demonstrate roentgenologically.

When the focus is seen at operation it appears as a definite cavity filled with brownish tissue. In the center is sometimes a well-ossified portion which may be mistaken for a sequestrum. The whole lesion is clearly demarcated from the surrounding hypertrophied bone.

Microscopic examination reveals a background of very vascular fibrous tissue, in which may be seen giant-cells. In this stroma are masses, strands, or trabeculae of osteoid tissue which, in its older portions, is irregularly calcified to form immature bone. There is never any evidence of hemorrhage or infection.

The treatment of osteoid-osteoma is most satisfactory for immediate and permanent relief of symptoms following excision of the nidus. It is not necessary to remove all of the hypertrophic regional bone, which may gradually resorb, particularly in children, but if the nidus is not excised completely, persistence or recurrence of symptoms is the rule.

Since of 158 patients there were only 11 who were older than 30, one is justified in assuming that the lesion may heal spontaneously. In this connection the history of a man who is not included in this series is of interest. In 1916, when he was 18 years old, he consulted

Dr. D. B. Phemister because of pain, tenderness, and swelling which had been present in the lower left tibia for one and one-half years. Roentgenograms showed a lesion which we would now have no hesitancy in calling an osteoid-osteoma. Surgery was refused by the patient. Gradually the pain and tenderness decreased, and by 1921, seven years after their onset, they had disappeared and did not return. In 1940, 24 years after his initial visit, the patient was examined again. Swelling was still present but there was neither pain nor tenderness. A roentgenogram shows persistent sclerosis and thickening of the cortex, but no sign of the nidus.

The etiology of these lesions is still unsettled and is the subject of much discussion. Most authors who have studied actual examples of osteoid-osteoma are in agreement with Jaffe that it is a distinct entity. There exists, however, no such agreement as to whether the lesion is, as Jaffe contends, a benign neoplasm.

Previous trauma is frequently mentioned as a possible predisposing factor, as it is with almost any skeletal lesion. Among our thirty patients there were three with a history of severe trauma to the hand or wrist who stated that symptoms had begun immediately and had never since subsided. Of these, two had lesions in the phalanges of the fingers and one in the distal radius. Five other patients gave a varying history of injury at various time intervals preceding the onset of trouble. Twenty-one patients stated specifically that there had been no trauma. In view of the known propensity of patients to relate all skeletal disorders to some type of injury and of the small number of such histories related to osteoid-osteomas, it seems safe to disregard trauma as of etiological significance.

The only authors who have denied categorically the existence of osteoid-osteoma are Brown and Ghormley. They reported a series of twenty-four patients whom they considered to be examples of the condition described by Jaffe. Ten of their patients were operated upon, and pathologic examination of the material removed showed "chronic inflammatory tissue." The photomicrographs presented certainly substantiate this diagnosis and do not at all resemble the picture of an osteoid-osteoma. In two cases there was cultured staphylococcus aureus and in two a micrococcus. The authors quite rightly called these cases bone abscesses. However, their reasons for maintaining that osteoid-osteomas are all identical solitary chronic bone abscesses are not very clear.

In a paper published in 1942, Brailsford presented several cases which had x-rays that are compatible with a diagnosis of osteoid-osteoma. From three of the patients who were operated upon staphylococcus albus was cultured. Because of this and because of the x-ray appearance, Brailsford calls these lesions chronic subperiosteal abscesses. He then makes a general statement that the pathology was like that described by Jaffe, but there are not enough data in the paper to be certain of the similarity. Because of this he assumed that all osteoid-osteomas are a distinctive variation of an infectious process.

Actually the weight of evidence is clearly against infection as the etiological agent in osteoid-osteoma. The patients never have fever, leukeocytosis, or other systemic manifestations. When swelling is present it is only occasionally warm, and there is no redness. The lesions are always solitary, never recur after complete excision, and after operation invariably heal per primum. The few positive cultures which have been reported have usually been of staphylococcus albus or some other common contaminant. In our own series cultures were made in twenty-two instances. Of these, nineteen produced no growth. One each produced streptococcus viridans, staphylococcus albus, and diphtheroids, all of which are considered to be contaminants. In five cases guinea pigs were inoculated and none of these showed evidence of disease. In six additional cases, skin tuberculin tests were negative. Twenty-four of the patients had negative Wassermann or Kahn examinations. It is clear that the history, physical findings, and course of these patients are not those of infectious processes.

If additional proof were needed, it is amply furnished by the constancy of the distinctive pathologic findings. In no case has there been found evidence of either acute or chronic infection. The lesions are always sharply demarcated from the surrounding hypertrophied regional bone. The bone immediately surrounding the nidus might possibly be confused with the bone adjacent to an abscess because the spaces are filled with fibrous marrow which often contains scattered foci of round cells.

This picture does not indicate infection as it is . in the reactive zone about many different s of lesions. It is also to be noted that the :al tissue of an osteoid-osteoma has never . seen in conjunction with a proven abscess . in those relatively indolent small abses which sometimes occur in patients multiple foci. Osteoid-osteoma are al- ; composed of the same few elements a :ground of vascular fibrous stroma conng multi-nucleated giant cells and osteoid ie which is irregularly calcified. The les differ from one another only in the pat- in which these components are arranged. r are not related to activity of the hemaietic tissue, for they occur with no appar- ;electivity in fatty and in active marrow, .e cortex, and even just beneath the periosteum. To date no one has found an osteoid-osteoma in scapula, clavicle, or cranial bones. Only one case has been seen in which the lesion was located in an epiphyseal ossification center.

Osteoid-osteomas, unlike other benign tumors, are never larger than a centimeter or so in diameter. Once they have reached a mature state, they do not grow although the hypertrophied regional bone may increase to form a huge mass. Furthermore, it is not only unusual for benign tumors to be painful, but the pain of osteoid-osteoma is out of proportion to its small size.

Nevertheless, in consideration of all the facts, we agree with Jaffe that osteoid-osteoma is probably best interpreted as a benign tumor.

X-RAY DIAGNOSIS

Edward L. Jenkinson, M.D.

The x-ray diagnosis of bone tumors is difult and often hazardous. From our experi:e it has been impossible to differentiate :se tumors histologically, that is, to the type cells from the x-ray findings. If the radiolist is able to tell the surgeon or the referig physician whether or not the tumor is be;n or malignant, I believe that he has done that is expected of him, and often this is ry difficult to do. The most important point far as the patient and referring physician e concerned is whether or not the lesion is ilignant. There are salient points that can be inted out on x-ray films which are helpful in arriving at a diagnosis, such as invasion to the bone, resistance of cartilage to malignant tumors, etc. Roentgenological therapy of primary bone tumors in our hands has not been satisfactory. With a few exceptions, most of the patients whom we have treated, who were inoperable, have received very little benefit. It is important in treating patients to have a histological diagnosis, as some tumors respond well to small doses of irradiation, and if given large doses, more harm than good is done. The most beneficial results are obtained in treatment of metastatic malignancies.

Course No. 2

WRIST AND HAND

Lecturer

Sterling Bunnell, M.D., Some Points in Reconstruction of the Hand

SOME POINTS IN RECONSTRUCTION OF THE HAND

Sterling Bunnell, M.D.

CONSIDERABLE PROGRESS in the reconstruction of hands was made by Army surgeons, who were faced with the vast task of attempting to restore function to about 20,000 hands. The work was done in nine different general hospitals to which this type of work had been assigned by Norman T. Kirk, Surgeon General, U.S.A. Many methods were tried and evaluated. Excellent results were obtained by many different officers who restored useful function and thereby demonstrated the practical value of reconstructing hands. A brief digest of the principles used is given below.

It was clearly demonstrated that best results were achieved not by passing the patient from one tissue specialty to another, such as plastic, neuro, and orthopedic, but by considering the hand as an anatomic or regional specialty like the eye, ear, nose, or throat. The problem could then be handled as a whole by one man versed in the many complicated intricacies and special considerations of the region. Each aspect of the problem overlaps and is interdependent with the others, so none can be properly attended to without mastering the problem as a whole. The hand, I am convinced, should be considered as an entity or specialty. Only by special training and study of this region can one reconstruct it adequately.

It may be asked, from the standpoint of organization in institutional or private work, what should be considered a hand, and what are its limits. Though palmistry stops at the wrist, the hand as a mechanical unit extends at least to the elbow. The forearm bones and muscles control the hand. From a dynamic standpoint the control of the hand starts from the opposite cerebral cortex. Arm and plexus nerves are hand nerves, so damage of them cripples a hand just as much as if the nerves were injured at the wrist.

It is the hand that is the important part of the upper extremity. The main use of the arm is to support and innervate the hand, placing and controlling it for function. The hand problem has many aspects and cannot be studied in a few months. The hand has been a rather neglected field of surgery, a blind spot in orthopedics, not from any lack of importance but because it presents a special and complicated problem.

Commencing with the early treatment of injury, the wound should be excised and covered at once either by skin grafting, swinging adjoining skin, or applying pedicle skin in the primary operation. If a wound remains open, the hand stiffens. The hand should be kept in the position of function, that is, with the wrist somewhat dorsiflexed, the proximal finger joints in flexion, and the thumb in opposition. Casts should stop at the distal crease in the palm, should have the thumb free for opposition, and should not immobilize any digits except the injured ones. Fractures should be set early, using traction - usually skin, but pulp or skeletal when needed - and always pulling around a curved splint in the position of function, never by tongue blade or banjo splint. A dislocated joint not reduced early, but after

two weeks, stiffens. From the time of injury, all parts of the hand which are safe to move should move and be kept moving until the time of return to work Hands stiffen from immobility, swelling, and open wounds.

In planning the reconstruction, the first step should be excision of the cicatrix to liberate tissue for nutrition and supply good cover by pedicle skin. Joints should be drawn into positions of function, and the bones should be aligned for proper mechanics. Repair of nerves and tendons follows. The primary requisite is to obtain sensation and prehension If these are present, no hand is so badly crippled that it is not preferable to an insensitive prosthesis

Skin

In supplying cover, free grafts are useful for burns without deep damage or to close areas from which flaps are swung Pedicle skin brings nutrition, allows movement of parts beneath, and stands wear. It is usually applied as a flap in one procedure from the abdomen raised in the direction and path of the thoracico-epigastric vessels and with a broad, flared base. The length may be 2 1/2 to 1. Open pedicles are unsuited, as hands stiffen from absorption. All raw surfaces should be closed by skin grafting, tubing, or making the pedicle stemless. Tube pedicles are preferred for irregular areas or to carry a broad skin flap. Small ones are closed by undermining, but thick pedicles are closed by skin grafting. Elastoplast or adhesive holds the hand firmly in place for three weeks, but the addition of a plaster of paris body cast insures against any mishap.

Flexion contractures draw the skin tight to themselves in every direction Beneath this is the deep cicatrix binding the lymph and blood vessels and the nerves, drawing all the tissues into a firm mass This prevents nutrition of the hand. The felxion contractures may cover only one third of the circumference of the wrist. The remainder of the skin, however, is pulled tight, and the deep cicatrix binds all the contents of the wrist. When we consider that all the lines of supply to the hand come through the slender wrist, it is clear that the nutrition of the hand will be very poor Our procedure should be to excise the surface cicatrix and undermine the borders of the skin to allow it to retract to its normal tension. The defect is then covered by a pedicle skin graft. In the second operation on the deep structures, the deep cicatrix is excised en bloc, as cancer is removed. This liberates all the structures of wrist and hand. They will then open up, breathe, and bloom again. The tissue will become limber, vascular, and of more normal appearance.

In order to prevent the scar from forming keloid and flexion contractures, all borders of pedicles should be carefully placed so as not to coincide with directions of push and pull, i e., they should not cross flexion creases at or near a right angle. As the hand is a movable organ, scars must be zigzagged, darted, and placed so as not to check any movement. The open skin of a pedicle expands but scars shrink. It is more important to give attention to the borders than to try to supply ample skin, which carries with it an excess of parasitic fat. Such dome pedicles are grotesque and need two operations to flatten them.

Joints

A big problem in the hand is stiffening of joints, especially in the position of nonfunction. This position is assumed if the key joint, the wrist, is allowed to remain flexed. The extensor tendons are then tightened and the proximal finger joints hyperextend and cannot flex. The thumb is drawn back to the side of the hand, and the fingers secondarily claw. From swelling and immobility the ligaments become short and thick. From precipitation of fibrin and proliferation of fibroblasts all becomes sealed and firmly organized. Both tendons and joints participate in this general sealing and congealing of the tissues. The joints of the hand are so accurately fitted together that slight contracture of ligaments stops motion.

When a hand stiffens in the position of nonfunction, movement of thè digits is useless, but if the joints are first drawn into a position of function, the little movement present, even if slight, is useful in picking up objects. The hand will then be used, and by use will continue to improve. A set of elastic or spring splints* has been developed to draw the hand into the position of function, and at the same time loosen and exercise it. These splints

*J Bone & Joint Surg 28 732 (Oct , 1946) Splints obtainable from H Weniger, 143 Valencia St , San Francisco, California

mobilize the hand. Rigid splints make rigid hands.

Unyielding joints require capsulectomy or arthroplasty. These procedures are very successful in metacarpophalangeal joints, but not very successful in interphalangeal joints. In capsulectomies, collateral ligaments are excised beneath the aponeurotic hood and (as also after arthroplasties) flexion of the joints must be maintained by leather slings and elastic bands until flexion is well established. The flexion so gained will be lost if the intrinsic muscles are not present to maintain it unless some other provision is made. For example, the sublimis tendon may be transferred from the palm to the lateral band of the dorsal aponeurosis, or the proximal pulleys of the finger may be advanced to give proper leverage to the long flexors, namely, increased angle of approach. Arthrodeses in position of function are especially useful in the base of the thumb and the wrist.

Bones

Malunion of bones, like dislocations or flexion contractures, so angulate a limb that muscle balance is upset from there distally, resulting in deformity. It is necessary to correct this skeletal alignment for proper mechanics of the tendons and the joints. Bones are realigned by rotary angulatory osteotomies. Rotation is checked by the plane of the nail and by determining whether the digits passively flex toward the scaphoid tubercle.

Defects in metacarpals and phalanges are filled sometimes with cortical bone from ulna or tibia but preferably with cancellous bone from rib or ilium. Cancellous bone heals in five weeks, whereas cortical bone takes two or three months. In all bone carpentry the pieces are fitted accurately together and immobilized by pinning from various directions with Kirschner wires. A small chip of bone, as a key graft, or firm pinning are necessary to insure union.

In the absence of a central metacarpal head, the remainder of that ray is removed and the marginal metacarpal, whether of index or little finger, is moved over into its place. This narrows and relaxes the hand, and by placing the metacarpal heads together, braces them, preventing rotation of the digits. Injury to a metacarpal may so damage the important soft parts surrounding it, intrinsic muscles, nerves, and tendons, that resection of the ray is preferable to reconstruction. If it is a central ray, the marginal ray is jogged over. The finger may be filleted, preserving its nerves, and laid on the hand to fill a defect.

Nerves

For a long time it has been established that repair of the small nerves of the hand as far as the distal crease in the finger and including the motor branches of the median and ulnar nerves is practical, easy to perform, and that it yields exceptionally good results.

A large gap in the nerves in the palm can be overcome by slitting down the motor bundle so as not to pull on it, and then, after freeing the main nerves and flexing the wrist or elbow, by drawing and suturing them to the distal fragments.

A gap as great as five inches in any of the three arm nerves has been successfully overcome by extensive freeing of the nerves through multiple incisions up the arm, flexing joints if necessary from neck to hand, and by transplanting certain nerves to span the joints. Letting out of the joints is not started until after one month. Then it is accomplished slowly, by snubbing instead of splinting, so that the patient may always relieve any ache from painful nerves by flexing his joints voluntarily. In this way ischemia of the nerves will not persist long enough to cause necrosis, as the patient himself will relieve the lack of vascularity.

Nerve grafts of small calibre, as in the hand and the face, are successful, as they are nourished by surrounding lymph through and through. Larger nerve grafts necrose in the center. A cable graft of several strands of sural nerve to bridge an arm nerve is also successful as each strand is nourished through and through. This principle seems very simple, but it is the secret of success in nerve grafting.

Injuries of plexus or arm nerves cripple the hand so terribly that several procedures are urgent. One is to repair the nerves as soon as possible, even before bones or joints are repaired. From the time of severance, the muscles supplied by them undergo fibrous degeneration, as do also the lower fragments of the nerves, making nerve repair useless unless done early. Another necessity is to prevent the hand from stiffening in the deformity

of disuse. The hand should be kept in the position of function, preferably by light spring splints. The joints and the moving structures should be kept mobile by physiotherapy, best applied by manipulation with the other hand.

In muscle imbalance from paralysis of only one or two nerves it is wrong to splint rigidly with the hand in the position opposite to that of the deformity. All that is necessary is to hold the hand in a position of function by use of spring splinting just strong enough to counterbalance the lack of the paralyzed muscles, the splints being small and light enough to leave the hand free for use.

Tendons

Repair of tendons was discussed in the 1943 volume, but mention should now be made of various tendon transfers to establish muscle balance. For radial palsy a tendon should be used to stabilize the wrist in dorsiflexion (pronator teres to extensor carpi radiales), and another to stabilize the base of the thumb in extension (slip of extensor carpi radialis to abductor pollicis longus or palmaris longus or flexor carpi radialis to same). Some tendon should be left to give sufficient flexion to the wrist to prevent its being off balance in dorsiflexion. The palmaris longus or flexor carpi radialis transferred to the abductor pollicis longus will furnish flexion to this wrist, or one of these tendons may be left in place.

The thumb opposes well after tendon transfer if the following principles are observed. The tendon must insert on the ulnar side of the base of the proximal phalanx, must pass directly over the metacarpophalangeal joint and then subcutaneously over the thenar eminence and be made to pull in the direction of the pisiform bone. Any muscle, tendon, or construction of pulley may be used to suit the case.

In paralytic claw-hand for ulnar and median nerve palsy several types of operations are available. The thumb may be adducted and the metacarpal arch curved by the tendon-T operation. In this operation the crossbar of the T is a tendon graft spanning between the adductor tubercle of the proximal phalanx of the thumb and the metacarpal of the little finger, having been passed behind the flexor tendons. The upright of the T is any long flexor tendon passing to the center of this cross piece. When pulled the T changes to a Y, adducting the thumb and curving the metacarpal arch

In ulnar palsy the pinch is poor, since the index finger cannot abduct against the thumb and the thumb cannot adduct against the index. Much improvement in pinch or function is gained by transferring a tendon to substitute for the first interosseus muscle. For this there is the extensor indicis proprius, the extensor pollicis brevis (Bruner), or flexor sublimis of index of other finger. The sublimis may be passed in back of the thumb (Graham).

Replacing Digits

After loss of a thumb we are fortunate if part of the metacarpal is present because this will give movement. We can base on it either an adjoining finger or a tube pedicle with a bone graft.

It is very important to bring normal nerve supply to the newly constructed thumb whenever possible. When the index finger is pollicized nerves should be brought with it. Also, all tendons should be connected up for movement.

When the tactile part of a thumb or finger has been replaced by pedicle skin it is advisable to make an exchange of skin flaps between it and the adjoining tactile skin. This will restore sensation to the digit where needed.

If the metacarpal of the thumb is gone, a synthetic post can be made by tube pedicle and bone graft into carpus preferably into trapezium. It should be short and thick and must be placed exactly in the zone of motion of the fingers, so that they can either pass it or work against it If it is too long, it may break, if wrongly placed, it may be useless.

When only a few digits are left in a hand, these, by rotary angulatory osteotomy, should be placed to oppose each other for prehension. If all digits have gone, distal to the metacarpals, much function can be regained by phalangizing the thumb and by removing the second and fourth rays, thus creating a three-digit hand. By osteotomy, the outside digits can be made to oppose each other.

Course No. 3

PHYSIOLOGY OF BONE

Lecturers

Franklin C McLean, M D., Introductory Remarks - "The Physiology of Bone"

Edward C. Reifenstein, Jr., M.D., "Effect of Steroid Hormones in Osteoporosis"

W. D. Armstrong, M.D., and Edmund P. Flink, M.D., "Interpretation of Laboratory Data in Skeletal Disorders"

INTRODUCTORY REMARKS

Franklin C. McLean, PhD., M.D.

THE PRESENCE of so many listeners at a symposium entitled "The Physiology of Bone" is perhaps in itself an answer to the question Why should orthopedic surgeons concern themselves with the more fundamental aspects of the physiology of bone? Their presence at such a symposium indicates that they are interested, and that is sufficient justification - if justification were needed - for devoting an entire afternoon to matters that may seem to be far removed from the practice of orthopedics.

I am devoting a few paragraphs to my conception of the function of the orthopedic surgeon, from which it may appear why I, who have never had anything to do with orthopedic surgery, am rash enough to take part in a symposium such as this. First, I would like to indicate that I believe that the old-fashioned concept of the skeleton as purely mechanical framework for the muscles and the viscera is gone forever. And with it has gone the idea that the orthopedic surgeon is merely the carpenter or the structural steel worker who tinkers with the framework. Orthopedic surgeons will continue to find plenty of mechanical problems, but it is my hope and expectation that their horizons will extend far beyond this aspect of their work.

Few of those who are in training in orthopedic surgery today are old enough to remember that World War I marked the transition from the idea of rigidity, as applied to the skeleton, to the idea of plasticity. The realization that bone is a plastic tissue, which can be bent to the will of the surgeon, did not come into full bloom until after the first World War, and in part as a result of the lessons learned during that war. This idea is now a commonplace to orthopedic surgeons, and perhaps not all of them are fully aware of how completely its acceptance has transformed the specialty into which they have entered or are about to enter.

I believe that an equally important transition is now in process. While it would be wrong to attribute this transition to World War II, it will probably turn out that many of those who have emerged from the second World War with their eyes on the future of orthopedic surgery will play large parts in the establishment of new points of view.

It might be that some will object to my description of the future function of the orthopedic surgeon, on the ground that what I have to say has little or nothing to do with surgery. But I refuse to be bound by a narrow definition of surgery or of the surgeon. To me surgeons are physicians first of all. They become designated as surgeons because they make large use of the operating room and of certain techniques, but their primary concern is with

disease and deformity rather than with these techniques.

With the idea that bone is plastic has come a realization that bone is also a tissue and that it undergoes a long period of growth and development, during which it is subject to many influences, nutritional, hormonal, and environmental. And even when growth is complete the skeleton is not at rest. I shall not dwell on traumatic accidents, or upon tumors or infectious disorders of bone, for enough emphasis will be given to these by others. But there are such systemic disturbances as hyperparathyroidism, Paget's disease, and postmenopausal osteoporosis, to say nothing of the osteomalacia widespread in a war-ridden world.

My question then becomes Who is to accept the challenge of the nontraumatic disorders of the skeleton? And my answer is that you will. And you will for the simple reason that in the long run no one will know more about the bones and their idiosyncrasies than you will. Wherever you are in practice you will find that these problems will be laid on your doorsteps, and that your success in practice will be measured not only in terms of your ability to deal with injury and deformity, but equally in terms of your grasp of the metabolic and systemic problems that come to you.

And even if you were to reject the idea that nontraumatic disorders of the skeleton belong in your province, you cannot escape the influence of systemic factors upon the healing of accidental injuries to bone and of the injuries that you yourselves produce by your operative approach to the skeleton. Without some idea of the behavior of bone you will constantly be plagued with doubts as to the course of events in your dealings with your every-day problems.

The field that I am outlining as being within your province may be called that of the normal physiology. And it is a truism that, in order to understand pathological physiology, one must know physiology The internist accepts this principle in his dealing with the pathological physiology of carbohydrate metabolism, you will find it essential to a grasp of the abnormalities of calcium metabolism and of the pathological physiology of bone

At this point it would be useful if I could refer you to a comprehensive monograph or treatise on the physiology of bone, including the deviations from the normal, upon which you could base your further explorations in the field. But the simple fact is that no such a volume exists. And even the writers of textbooks of physiology are hardly aware of the fact that bone has a physiology, demanding treatment on a par with that of the liver and kidneys. A recently published textbook of physiology of well over a thousand pages gives less than a page of material relevant to bone. These facts are additional evidence for the need for such a transition period in your specialty as I see in progress, and I confidently look to this generation to correct the deficiencies both in knowledge and in its application.

This program has been arranged by the Committee of Instructional Courses as illustrative of the problems presented by the physiology of bone. We cannot hope to give you a complete or even a wholly systematic course in this wide field within the limits of an afternoon. But if we can give you an introduction into this field perhaps you can go on from there. And this may possibly lead to some reorientation in future programs of such societies as this. I have looked through the program for this six-day session of the American Academy of Orthopaedic Surgeons and find almost nothing, except for this afternoon's session, within the field which I have outlined as falling within your province. Perhaps the time will come when a fair proportion of the scientific program of the society will be devoted to systemic and metabolic disorders of bone I look to you to bring about this much of a shift in emphasis

Today's program has been built around the papers which will be presented by Dr. Reifenstein and Dr. Flink. Dr Armstrong was to have discussed the interpretation of laboratory data in skeletal disorders but unfortunately became ill at the last moment, his place will be taken by Dr. Flink, and their contribution will be published under their joint authorship. Dr. Reifenstein will discuss the effect of steroid hormones on osteoporosis, a subject of great and still increasing interest. I shall devote the remainder of my period to a brief review of some work on nutrition, mineral metabolism, and vitamins in relation to bone. Since most of what I have to say was published in some detail in last year's volume, entitled *Regional Orthopaedic Surgery and Fundamental Orthopaedic Problems, Selected from the In-*

structional Courses of the Thirteenth Annual Assembly of the American Academy of Orthopaedic Surgeons, Chicago, January 19-23, 1946, pp. 110-119, as published by J. W. Edwards, Ann Arbor, Michigan, I shall not include this material in my published remarks. I shall, however, publish a few paragraphs in amplification of the papers by Drs. Reifenstein, Armstrong, and Flink.

First, in both of the papers to be published herewith there is a considerable amount of discussion concerning phosphatase and its special relationships to bone. The idea has become firmly fixed in the literature, and is repeated by Dr. Reifenstein, that phosphatase plays a role in the calcification of bone by increasing the local concentration of phosphate ions, by the splitting off of these ions from organic phosphate compounds, and thereby premoting the deposition of the calcium-phosphate-carbonate complex commonly known as "bone salt."

This very popular theory ignores the facts that certain tissues which are not ordinarily calcified - namely, the intestinal mucosa and the kidneys - are equally rich in an alkaline phosphatase identical with or closely resembling that found in bone, and that no one has as yet been able to find the organic phosphate compounds from which the inorganic phosphate ions are presumed to be split off. The presumption of a relationship of phosphatase to calcification therefore rests upon circumstantial evidence. Phosphatase is generally present where calcification is occurring, and it has chemical properties which could be useful in promoting this process. That it actually plays a part in this process, however, remains to be shown.

Without going in detail into any alternative ideas, let me ask that you keep your minds open as to the role of phosphatase in bone. There is no doubt, as Dr. Flink will tell you, that there is a close correlation between blood phosphatase levels and bone formation, as indicated microscopically by osteoblastic activity. The correlation can also be shown by direct observation of the phosphatase in bone, and it may be accepted as demonstrated that osteoblasts engaged actively in the formation of bone are rich in phosphatase. There is, however, good evidence that osteoblastic activity has little or nothing to do directly with calcification. And there are reasons, which cannot be gone into here, for believing that the phosphatase of the osteoblasts is concerned with the formation of the inorganic bone matrix, rather than with the calcification which follows after the matrix is laid down.

In the laboratories of Dr. Bloom and myself, at the University of Chicago, we have intensified our interest in the effects of estrogens on bone. Our earlier work was reported in last year's volume, above referred to. Dr. Reifenstein's paper reports in some detail on the effects of estrogens on the human skeleton. We are currently concerned with observation of dosages and of species differences in the response of mammals to estrogens, our interest being stimulated by the fact that these hormones are the only ones which seem to have a direct effect in the promotion of formation of new bone tissue. Attempts have been made by others to utilize this effect in the treatment of experimental and of accidental fractures, with equivocal results. It now appears that the dosages necessary to influence bone production are in general in a higher range than the maintenance doses required in the postmenopausal state, so it may perhaps be necessary to repeat some of the earlier work with the use of larger doses.

The effects of estrogens on the bones of mice are very similar to those in birds, in which the effects were first described. In the rat, however, as stated in last year's volume, the increase in medullary bone in the growing animal is attributable to failure to resorb the spongy bone formed in the process of endochondral ossification. The result is a picture very closely resembling that in marble-bone disease, although no etiological relationship between the two conditions has been established. One may await with interest the developments of the next few years in this field.

EFFECT OF STEROID HORMONES ON OSTEOPOROSIS*

Edward C. Reifenstein, Jr , M.D.

Normal Bone Metabolism

Normal bone is composed of an organic matrix in which is deposited a calcium-phosphate-carbonate salt. It has three types of surface - one on which nothing is happening, one on which bone is being resorbed, and one on which bone is formed (Fig. 1). Where bone is being resorbed, one sees osteoclasts. Bone formation consists of two steps, the laying down of the matrix by osteoblasts, and the deposition therein of the calcium salt.

It should be emphasized that bone formation and bone resorption occur at the same time. The reason why the body can deposit calcium on one surface while it is being resorbed on another is probably connected with the enzyme phosphatase. It is thought that the enzyme is made by the osteoblasts, and its presence where bone matrix is being formed increases the local concentration of phosphate ions by the splitting off of these ions from organic phosphate compounds, and that it therefore promulgates the deposition of a calcium-phosphate-carbonate salt. In the absence of hepatic disease, the serum alkaline phosphatase level is an index of the activity of the osteoblasts. In the presence of excessive osteoblastic activity the serum phosphatase level rises, with osteoblastic inactivity it tends to fall. Since the normal level of the serum phosphatase is extremely low, however, this fall is not usually discernible.

There is considerable evidence that stresses and strains strongly stimulate the osteoblasts to lay down bone matrix, the greater the stresses and strains, the greater is the bone formation. Thus, persons whose occupations involve considerable physical activity have heavy skeletons, whereas those with sedentary occupations tend to have light bones.

Types of Osteogenesis

There are three types of bone formation (a) endochondral, (b) membranous, and (c) endosteal.

The steps in the formation of bone from cartilage are· (a) proliferation of cartilage cells with increase of intercellular cartilaginous substance, (b) arrangement of more mature cells into rows, (c) calcification of intercellular cartilaginous substance between rows, (d) the breaking of blood vessels into the lacunae containing the cartilaginous cells, (e) the laying down of bony matrix (osteoid) by osteoblasts on to the surfaces of the calcified cartilaginous trabeculae left after the blood vessels have broken into the lacunae, and (f) the deposition of a calcium-phosphate-carbonate salt into the osteoid.

The term "membranous bone" is usually reserved to designate that bone which is formed directly from specialized mesenchymal tissue in the embryo without there having been any preceding cartilaginous phase. I will use this terminology, perhaps somewhat loosely, to include periosteal bone formation.

By "endosteal bone formation" I refer to that appositional bone laid down in the cortex and trabeculae of bone as a part of the constant remodeling of bone which takes place during growth and after growth has ceased.

In the final analysis it will be noted that all three of these types of bone formation are essentially the same, namely the laying down by osteoblasts of an extracellular substance called osteoid, and the deposition into this osteoid of a calcium - phosphate - carbonate salt.

*The work described in this communication was done in the Medical Service of the Massachusetts General Hospital and the Department of Medicine of the Harvard Medical School in association with Dr Fuller Albright.--The expense of these studies was defrayed by grants from the Josiah Macy, Jr , Foundation, from the Rockefeller Foundation, and from the National Research Council (Committee for Research in the Problems of Sex) A bed supported by the Mallinckrodt Chemical Company on the Metabolic Ward was used for part of these studies

Bone Disorders Due to Hormone Abnormalities

Some of the most important bone disorders arising from abnormal function of the endocrine glands are listed in Table I. Since osteoporosis is the predominant pathologic condition I will devote the remainder of the lecture to this condition.

Table I

Bone Disorders Due to Hormone Abnormalities

Hormone Abnormality	Name of Disease	Bone Condition
Parathyroid Excess	Hyperparathyroidism	Osteitis Fibrosa Generalisata
Parathyroid Lack	Hypoparathyroidism	Decreased Bone Resorption
Thyroid Excess	Hyperthyroidism	Osteoporosis (? Indirectly)
Pituitary Growth Hormone Excess	Acromegaly	Osteoporosis (? Indirectly)
Androgenic Lack	Eunuchoidism	Osteoporosis
Estrogenic Lack	Post-Menopausal State	Osteoporosis
Adrenal Cortical "S" Hormone Excess	Cushing's Syndrome	Osteoporosis

Osteoporosis, Osteomalacia, and Osteitis Fibrosa Generalisata

Osteoporosis, osteomalacia, and osteitis fibrosa generalisata have one thing in common, a decreased bone mass. The total bone mass may be diminished to a pathologic degree from either an increase in bone resorption or a decrease in bone formation. The former is called "osteitis fibrosa generalisata" (Fig. 1) and is found in hyperparathyroidism. A decrease in bone formation may result either from an underactivity of the osteoblasts in laying down bone matrix or from a failure in the deposition of calcium salts in this matrix, the former is termed "osteoporosis," and the latter "osteomalacia" (Fig. 1).

Classification of Metabolic Bone Disease in Adults

Since the total mass of calcified bone may be changed in quantity either by altering the bone resorption or the bone formation, and since bone formation consists of the two steps in sequence of matrix formation and the calcification of the matrix, we may classify metabolic bone disorders in adults as in Table II.

Definition of Osteoporosis

It should be clear from what has already been said that osteoporosis is not synonymous with demineralization of bone; it is that category of too-little-bone where the primary disturbance is lack of bone matrix formation. It is not to be confused with osteomalacia, where the primary disturbance is failure of mineralization of bone, or with osteitis fibrosa generalisata, where the primary disturbance is increased bone destruction.

Conditions Associated with Osteoporosis

In clinical medicine one encounters the following conditions associated with osteoporosis. (1) disuse atrophy, where the normal stimulus to osteoblastic activity is absent; (2) old age, where the bone tissue like other tissue (cf. hair, skin, muscles) atrophies; (3) malnutrition, where the protein requirements are not fulfilled, and the bone matrix, like other tissues, is depleted, (4) Cushing's syndrome where, we believe, an excess of the adrenal cortical "sugar" or "S" hormone inhibits anabolism of protoplasm including bone matrix, (5) adaptation syndrome of Selye, where, we believe, the pathological physiology is the same as in Cushing's syndrome, (6) idiopathic osteoporosis, where the cause of the condition remains obscure, (7) acromegaly, where the cause may be the increase of pituitary hormone(s), or the secondary lack of gonadal hormones; and (8) the postmenopausal state, the commonest of all forms, where the difficulty is a deficiency in estrogen to stimulate the osteoblasts. Frequently 2 or more factors combine in one individual; thus, after an orthopedic operation factors 1 and 5 probably both play a part.

I have classified the conditions associated with osteoporosis somewhat differently in Table III. The term "Defect in Osteoblasts" is used to refer to those conditions in which the osteoblastic cells are defective, the term "Defect in Matrix" is used to indicate those conditions in which the osteoblastic cells are

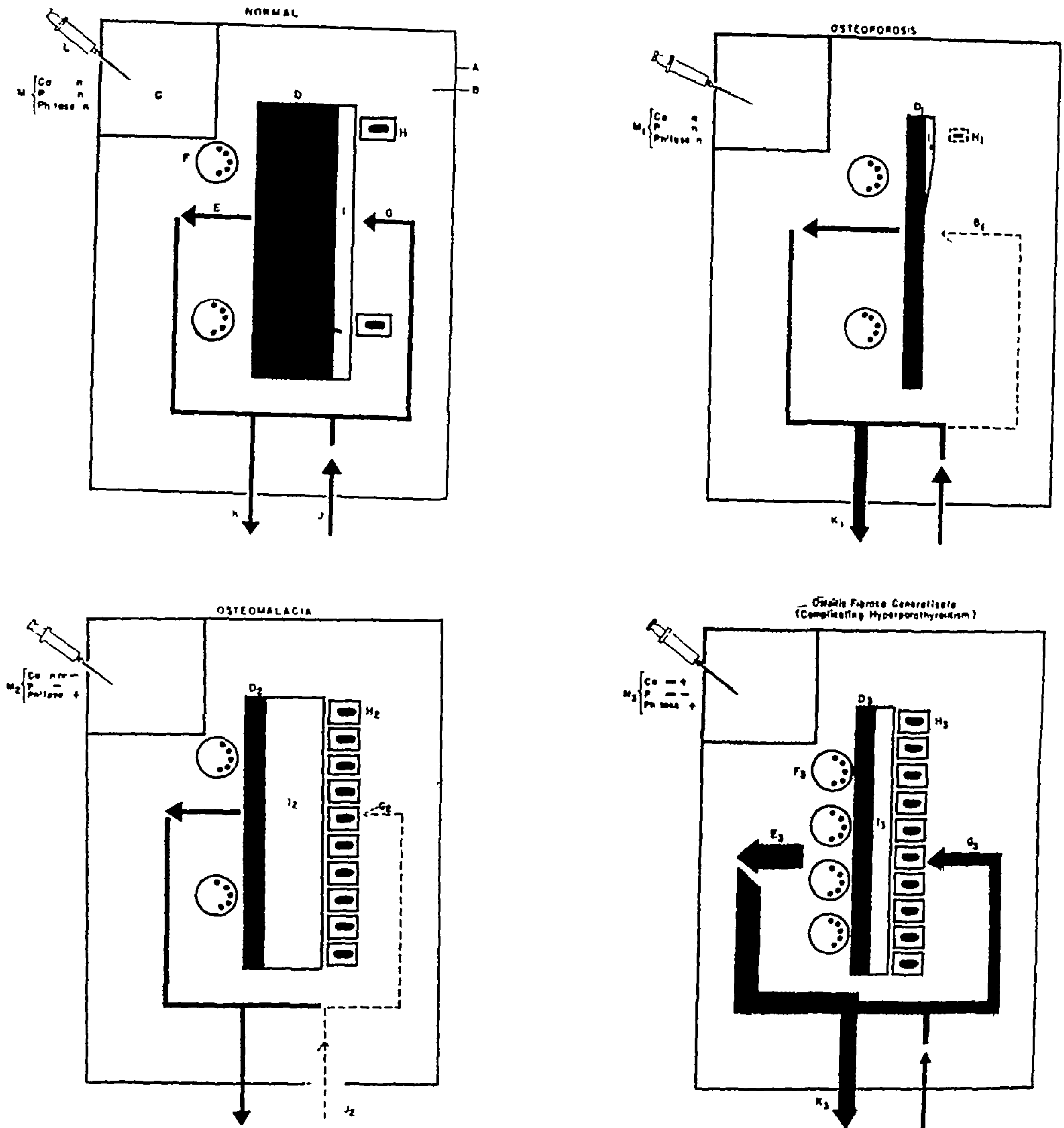


Fig. 1 Schematic Diagrams Showing the Differences in Bone Metabolism between Normal, Osteoporosis, Osteomalacia and Osteitis Fibrosa Generalisata Complicating Hyperparathyroidism (reproduced by permission of the Journal of the American Medical Association)

In all the diagrams, the designations are as follows A - body limits, B - body fluid, C - body serum, a compartment of body fluid easy to tap for analysis, D - bone mass with two surfaces, one where bone is being resorb d and one where it is being laid down, E - arrow indicating by its size the rate of calcium and phosphorus resorption, F - osteoclast, G - rate of calcium and phosphorus deposition, H - osteoblast laying down osteoid (I), J - calcium and phosphorus entering the gastrointestinal tract, K - calcium and phosphorus leaving the body by the kidneys and other exits, L - syringe for obtaining serum for analysis, M - blood values (N, normal, +, high, and -, low)

Table II

Metabolic Bone Diseases in Adults

I Too little calcified bone
- A. Bone formation too little
  - a) Defect in matrix formation osteoporosis
  - b) Defect in calcification of matrix osteomalacia
- B. Bone resorption too much
  - a) Osteitis fibrosa generalisata

II Too much calcified bone
- A Bone formation too great
  - a) Increased matrix formation elemental phosphorus poisoning, excessive stress, healed osteitis fibrosa generalisata, and healed osteomalacia
  - b) Increased calcification of matrix nonexistent
- B Bone resorption too little
  - a) Osteopetrosis
  - b) Hypoparathyroidism

Table III

Causes of Osteoporosis

I. Defect in osteoblasts
- A Loss of stress and strain
  - 1) Atrophy of disuse
- B Lack of estrogen
  - 1) Post-menopausal state
  - 2) Congenital hypoestrinism ovarian agenesis
- C. Congenital osteoblastic defect
  - 1) Osteogenesis imperfecta

II Defect in matrix
- A Loss of androgen
  - 1) Eunuchoidism
  - 2) ? Senile osteoporosis
- B Loss of protein
  - 1) Malnutrition
  - 2) Hypovitaminosis C
  - 3) Cushing's syndrome
  - 4) "Alarm reaction"

III. Defect unknown
- A Acromegaly
- B. Idiopathic osteoporosis

probably normal but lack the materials with which to form matrix.

Secondary Alterations in Calcium Metabolism in Osteoporosis

Osteoporosis being by definition primarily a disorder of tissue metabolism and only secondarily one of calcium metabolism, no alteration in the serum calcium and phosphorus levels is to be expected, nor is one usually found. Since in osteoporosis bone formation decreases and bone resorption continues unabated, hypercalciuria occurs, and may lead to the same kidney complications that are found in hyperparathyroidism, in which the hypercalciuria results from the hypercalcemia.

The degree of hypercalciuria, the diet being constant, depends on the discrepancy between bone destruction and bone formation. If osteoporosis develops gradually, this discrepancy is at no time great and hypercalciuria is not marked. If, on the other hand, bone formation suddenly stops in a nonosteoporotic skeleton, there is marked hypercalciuria, for example, in artificial menopause or immobilization of the skeleton in a cast. Furthermore, in young subjects, in whom the turnover of bone is more prominent than in older ones, a sudden curtailment of bone formation results in a marked imbalance.

Fig. 1 (Contd)

NORMAL. Note that the deposition of calcium and phosphorus (G) equals the resorption (E) and that part of the latter goes back into the bone.

OSTEOPOROSIS. Note the decrease in bone mass (D_1), the primary hypoplasia of osteoblasts (H_1), the decreased deposition of osteoid (I_1), the decreased deposition of calcium and phosphorus (G_1), the increased excretion of calcium and phosphorus (K_1), and the normal blood values (M_1).

OSTEOMALACIA. Note the decrease in bone mass (D_2), the hyperplasia of osteoblasts (H_2) because of increased stresses and strains, the increased deposition of osteoid (I_2) inadequately calcified because of the low serum calcium and phosphorus values, the decreased deposition of calcium and phosphorus (G_2), the primary difficulty in absorbing calcium and phosphorus from the gastrointestinal tract (J_2) and the abnormal blood values (M_2).

OSTEITIS FIBROSA GENERALISATA COMPLICATING HYPERPARATHYROIDISM. Note the increased excretion of calcium and phosphorus in the urine (K_3), the increased resorption of calcium and phosphorus (E_3), the increase of osteoclasts (F_3), the decreased bone mass (D_3), the increased bone formation by osteoblasts (I_3) because of increased stresses and strains, the increased deposition of calcium and phosphorus (G_3) because the serum is not undersaturated in respect to calcium phosphate, and the high phosphatase level (M_3).

Typical Course in Postmenopausal Osteoporosis

Five years after menopause, a woman develops pain in the back during rough automobile trip and is found to have spontaneously fractured vertebra. She may also have urinary complaints and be found to have kidney stones.

X-Ray Appearance of Bones

A word should be said about the appearance of osteoporotic bones in x-ray films. The bones are less dense rather than smaller. The involvement is most marked in the vertebrae and pelvis, next in the long bones, and then in the skull. The vertebrae may be crushed, wedged, codfish-shaped (like the normal codfish), or they may show herniation of the disc ("Schmorlsch Knötchen").

Metabolic Studies in Osteoporosis

A. Preliminary Discussion -- A detailed discussion of the methods employed in the accumulation, interpretation, and presentation of the data of the metabolic studies is beyond the scope of this communication, and will be found elsewhere.

In general the patients were placed on a metabolic ward and given a constant intake, which was analyzed. All urine and stool specimens were collected and analyzed for nitrogen, phosphorus, calcium, and at times other minerals. Frequent determinations were made of the levels of various chemical constituents of the blood, hormone assays were done as indicated. Fluid intake and body weight changes were recorded.

Precautions were taken to maintain the regimen as constant as possible so that the only addition to the program was the steroid hormone preparation being tested. As a check on the accuracy of the data obtained certain calculations were made. These were based on two assumptions 1) All of the body phosphorus, for practical purposes, is contained either in bone (calcium to phosphorus - 2.23) or as an integral part of protoplasm (nitrogen to phosphorus ratio of muscle = approximately 14.7), and 2) certain other components of muscle protoplasm exist in a fixed ratio to the nitrogen of the protoplasm. The factors employed in deriving these components are given in Table IV. With these assumptions, one can calculate from the nitrogen balance the theoretical balance of the other constituents that must be present if the hormone therapy is affecting bone or protoplasm and

Table IV

Factors Used in Deriving Certain Components of Muscle Protoplasm From Nitrogen (N)

Factor Number	Component to be Derived	Final Unit	Factor +
1	Protein in protoplasm	gm	6.25
2	Protoplasm: fat-free but not extracellular-fluid free	gm	32
3	Protoplasm fat-free and extracellular-fluid-free ["true muscle" (50)]	gm	27
4	Intracellular fluid in protoplasm	cc	19
5	Extracellular fluid in protoplasm	cc.	5
6	Potassium in intracellular fluid in protoplasm	m eq	2.7
7	Sodium in extracellular fluid of protoplasm	m eq	0.77
8	Phosphorus in protoplasm	gm	$\frac{1}{15}$
9	Sulfur in protoplasm	gm	$\frac{1}{14.5}$
10	Fat calorically-equivalent to protoplasm	gm	2.8
11	Protoplasm (fat-free) minus fat calorically-equivalent to protoplasm	gm	29.2

+ Factor x nitrogen in grams = component

can then compare these theoretical values with the measured balances. Any gross errors in the data will thus be exposed.

That these assumptions are reasonably accurate is shown in Fig. 2 and Fig. 3. In Fig. 2 an analysis is made of the phosphorus, calcium, and nitrogen balances during a metabolic experiment in which marked fluctuations of the balances were induced by starting and stopping testosterone propionate therapy. This figure illustrates that the phosphorus balances can be very nearly accounted for by the calcium and nitrogen balances during both the control observations and the experimental periods. During the 22 five-day periods 29.1 gm. of phosphorus were retained; 14.2 gm. could be accounted for by the calcium retention, and 18.1 gm. by the nitrogen retention, thus, of the 29.1 gm. of phosphorus retained 32.3 gm. were explained by both the calcium and the nitrogen retention. The small discrepancy between the measured and the theoretical phosphorus balances would be eliminated by a 10 per cent error in the intake of calcium, phosphorus, or nitrogen This analysis supports the contention that nearly all the phosphorus retained as a result of testosterone propionate therapy is retained either as bone or as protoplasm. In Fig. 3 are charted A) the nitrogen balance resulting from a therapeutic agent (testosterone propionate), B) the theoretical nitrogen balance based on the potassium balance, C) the theoretical nitrogen balance based on phosphorus balance after the latter has been corrected for the calcium balance, and D) the theoretical nitrogen balance based on the sulfur balance. The close correspondence of the determined nitrogen balance and the three theoretical nitrogen balances is noteworthy. Thus, during the 14 five-day periods in which the patient received testosterone propionate there was a retention of 211.3 gm. of nitrogen, and the theoretical-nitrogen-balances-based-on-potassium, -on-phosphorus, and -on-sulfur, respectively were 217.6 gm., 173.1 gm., and 181.3 gm. This analysis supports the contention that testosterone propionate induced a retention of nitrogen, potassium, phosphorus, and sulfur in the proportions that exist in muscle protoplasm.

A word of explanation needs to be given about the method of graphic presentation of the data. The method of charting the metabolic data is shown in Fig. 4. It is constructed as

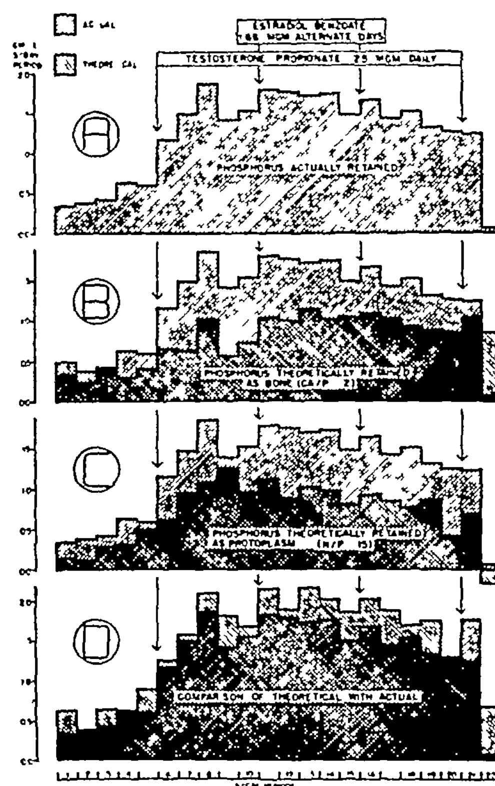


Fig 2 An analysis of the calcium, phosphorus and nitrogen balances in a metabolic experiment

This study on an elderly, male patient with osteoporosis (M H , M G H #267511) consists of 22 five-day periods The ordinate is the scale for balance in grams per five-day period, the abscissa is the scale for five-day periods The horizontal line starting at zero of the ordinate is the baseline, balances extending from the baseline toward the top of the chart are positive, those extending from the baseline toward the bottom of the chart are negative

The chart has four divisions A - the measured phosphorus balance, B - the measured phosphorus balance with superimposed theoretical balance explainable by the measured calcium balance (Ca/P = 2), C - the measured phosphorus balance with superimposed theoretical phosphorus balance explainable by the measured nitrogen balance (N/P = 15), and D - the measured phosphorus balance with superimposed theoretical phosphorus balance explainable by both the measured calcium and the measured nitrogen balances, i e , a summation of B and C. It will be noted in D that the actual phosphorus retention very closely follows the theoretical phosphorus retention As a matter of fact, if a ratio of Ca/P of 2 23 instead of 2 had been used in constructing the chart, the discrepancy would be very much less This experiment supports the contention that nearly all of the phosphorus retained as a result of testosterone propionate therapy is retained either as bone or as protoplasm

follows· a) the scale for intake and balance in gm./24 hr. is given as the ordinate; b) the scale for time (in this case days) is given as the abscissa, c) the horizontal line at O of the ordinate is the base line to which intake and balance refer, d) the intake is plotted as an area from the base line toward the bottom of the diagram, e) the excretion is plotted as a hatched area from the bottom of the intake toward the top of the diagram. If the excretion reaches the base line, the balance is in equilibrium. If the excretion exceeds the base line, a hatched area is left above the base line, this represents a negative balance. The data are plotted in amounts per 24 hours although the measurements are made on pools of excreta covering the metabolic periods. The fecal excretions are charted beneath, and the urinary excretions above the fecal excretions.

B. Metabolic Studies--

Case 1 Postmenopausal Osteoporosis, Artificial Menopause, Estradiol Benzoate Therapy.

F. F. (M. G. H. 156453), a 42-year-old woman, had a bilateral oophorectomy at the

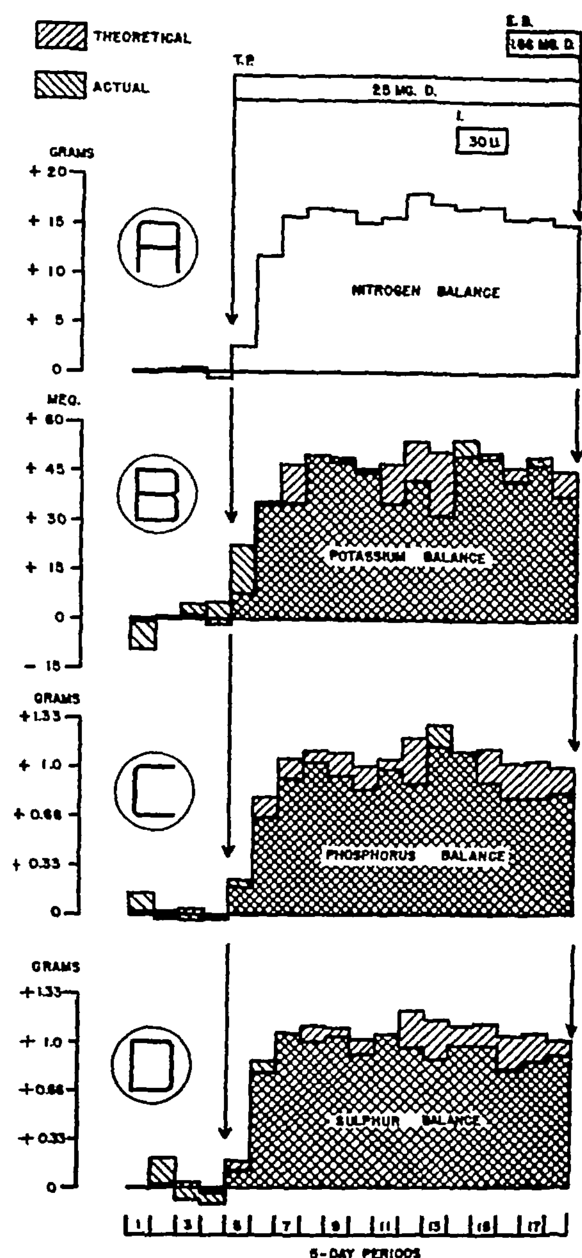


Fig 3 A comparison of the deviations in the nitrogen, potassium, phosphorus, and sulfur balances as a result of therapy

The subject of this analysis was a female patient with Cushing's Syndrome (B S , M G H #74372) The scales for balances in amounts per five-day period are given as the ordinates, the scale of five-day periods is given as the abscissa The analysis consists of 18 five-day periods The horizontal line starting at zero on each ordinate is the baseline of that particular balance, balances extending from the baselines toward the top of the chart are positive, those extending from the baselines toward the bottom of the chart are negative The balances are charted as deviations from the average of the control periods rather than as the deviations actually measured T P = testosterone propionate, I - insulin, E B = estradiol benzoate, D - dosage per day, U = units per day The data for potassium are based on analyses of urinary excretions alone, the fecal potassium excretion was assumed to be 8 per cent of the potassium intake The factors for the calculations are given in Table IV

The chart has four divisions A - the measured nitrogen balance, B - the measured nitrogen balance with superimposed theoretical nitrogen balance explainable by the measured potassium balance, C - the measured nitrogen balance with superimposed theoretical nitrogen balance explainable by the measured phosphorus balance (after the phosphorus theoretically retained with calcium had been subtracted as in Figure 2), and D - the measured nitrogen balance with superimposed theoretical nitrogen balance explainable by the measured sulfur balance. It will be seen that there is a close correspondence between the measured and the theoretical nitrogen balances This is evidence that testosterone propionate therapy induced a retention of nitrogen, potassium, phosphorus, and sulfur in the proportions that exist in muscle protoplasm

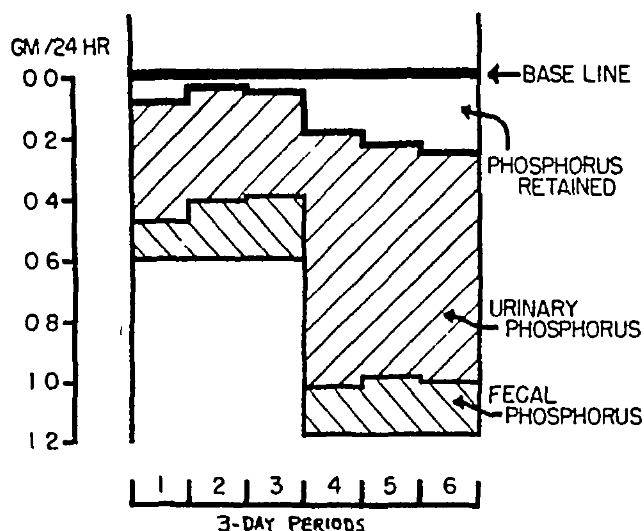


Fig 4 Method of Charting Metabolic Data.

age of 41 for endometriosis, following the operation she had "nocturnal seizures," the exact nature of which was not determined. During the following year there was a gradual onset of back pain with increasing dorsal kyphosis and a loss of energy. On admission one year after operation, the patient was in good physical condition except for the deformities of her spine; her blood pressure was 130/80. X-rays revealed typical codfish deformity of many of the dorsal and lumbar vertebrae, a collapse of some vertebrae, and anterior wedging of others. Laboratory studies: serum calcium 10.5 mgm. per cent, serum phosphorus 4.2 mgm. per cent, serum alkaline phosphatase 3.6 Bodansky units, serum total protein 7.3 grams per cent, normal glucose tolerance test, some hypoglycemia unresponsiveness in an insulin tolerance test, basal metabolic rate of minus 6, follicle-stimulating hormone test positive for 25 mouse units per 100 ml., and 17-ketosteroid excretion of 4.3 mgm. per 24 hours.

The metabolic data of Case I are shown in Fig. 5. The first part of the study, conducted in 5-day periods, consisted of: 1) three control periods, 2) five periods with estradiol benzoate 1.66 mgm. intramuscularly every 3 days; 3) twenty-three days with the same therapy at home; 4) two periods with the same therapy, 5) two periods with progesterone 10 mgm. intramuscularly daily in addition to the estradiol, and 6) twelve periods after the cessation of both medications. The patient was then discharged on estrogen therapy which was given continuously in varied dosage during the next 3 years; during this interval she was brought back to the metabolic ward for study (1 to 3 five-day periods) on 3 occasions.

The data (Fig. 5) are self-explanatory. Attention should be called to 1) nitrogen phosphorus, and calcium equilibria during the control periods (1 to 3), 2) the high serum phosphorus level which tended to fall under estrogen therapy (less marked in this case than in the others (see below)), 3) the slight improvement in nitrogen balance under estrogen therapy, 4) the striking and growing decrease in calcium excretion, both fecal and urinary, with estrogen treatment and the gradual return (40 days) in calcium excretion to pre-treatment levels following cessation of estrogen therapy, 5) a decrease with estrogen treatment in the phosphorus excretion almost entirely confined to the urinary component, and reasonably proportional to the changes in the calcium and nitrogen metabolism, 6) failure of the serum phosphatase level, the index of osteoblastic activity, to rise under estrogen therapy; 7) an increase in nitrogen, but not in calcium and phosphorus, excretions in periods 11 and 12 with progesterone therapy; and 8) the tendency to retain extracellular fluids with estradiol therapy, as suggested by the increase in the actual weight above the theoretical weight.

The apparent discrepancy in the effect of estrogen on the calcium and phosphorus balances during periods 26 and 27 is probably to be explained by erroneously high fecal excretions resulting from too short a period of observation.

Case 2: Postmenopausal Osteoporosis; Physiological Menopause, Question of Superimposed Atrophy of Disuse; Estradiol Benzoate Therapy.

E. P. (M. G. H. 203540), a 60-year-old patient, had a physiological menopause at 53. Thirteen months before admission she fell down six steps and fractured her first lumbar vertebra; she was kept in bed five months for this injury, and then allowed up with a brace. Eight months before admission the 9th dorsal vertebra collapsed. Except for back and chest pain, the patient had no complaints, and was in good general health upon admission. Her blood pressure was 120/90. X-ray examina-

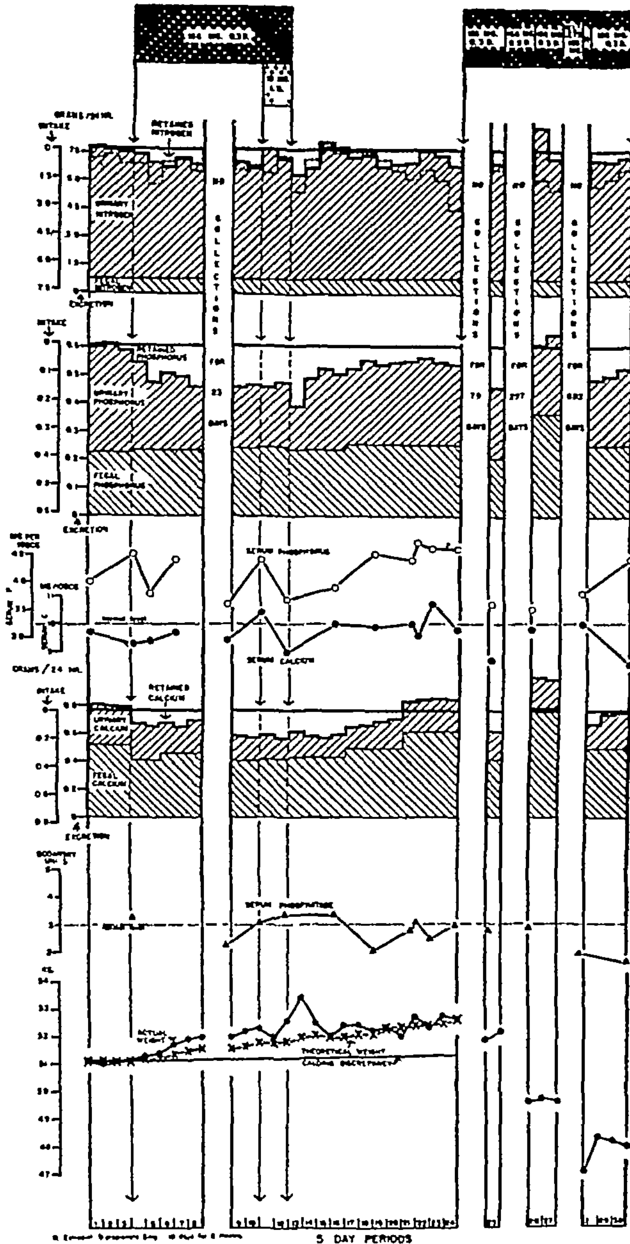


Fig 5 Case 1 (F F , M G H 156453) Effect of Extradiol Benzoate on Nitrogen, Phosphorus, and Calcium Balances, on Serum Calcium, Phosphorus, and Alkaline Phosphatase Levels, and on Body Weight in a Female Patient with Post-Menopausal Osteoporosis

For discussion, see text
The dotted line in the nitrogen metabolism data represents the "theoretical nitrogen balance " The fecal nitrogen was estimated as 10 per cent of the intake The fecal calcium and phosphorus values as charted are averages of 1, 2, 3, or 4-day periods as follows 1 through 3, 4 through 5, 6 through 8, 9 through 10, 11 through 12, 13 through 16, 17 through 20, 21 through 24, 25, 26 through 27, 28 through 30

tion revealed the fractures of the first lumbar and the 9th dorsal vertebrae, marked osteoporosis of the spine and pelvis, but not of the skull, and gall stones. Laboratory studies serum calcium 10.1 mgm. per cent; serum phosphorus 3.5 mgm. per cent, serum alkaline phosphatase 3.7 Bodansky units, serum total protein 7.6 grams per cent, no Bence-Jones protein in the urine.

The metabolic data of Case 2 are shown in Fig. 6. The study, conducted in 5-day periods, consisted of 1) five control periods, 2) thirteen periods during which the patient received estradiol benzoate 3.32 mgm. intramuscularly every other day. In addition, during the three periods 14, 15, and 16, testosterone propionate 25 mgm. were administered intramuscularly every other day.

The data in Case 2 confirm the main observations made on Case 1. The fall in the serum phosphorus level after estradiol medication was more pronounced than in Case 1, and in addition there was a fall in the serum calcium level. Again the serum phosphatase level failed to rise with the improvement in the calcium balance. The duration of the testosterone propionate therapy was too short to judge its effect on the calcium balance, it brought about the expected increase in the nitrogen retention and rise in the urinary 17-ketosteroid excretion. The theoretical nitrogen balance based on the phosphorus balance after it had been corrected for the calcium balance agrees quite well with the measured nitrogen balance.

Case 3 Postmenopausal Osteoporosis, Artificial Menopause, Methyl Testosterone, Estradiol Benzoate and Pregnenolone Therapy.

R. W. (M. G. H. 319940), a 56-year-old woman, had a cholecystectomy at 26, and thyroidectomy for thyrotoxicosis at 46. At 48, an artificial menopause was induced with radium for metropathia hemorrhagica. Three years before admission the patient strained her back opening a heavy window, and thereafter had several episodes of sharp pain in the back

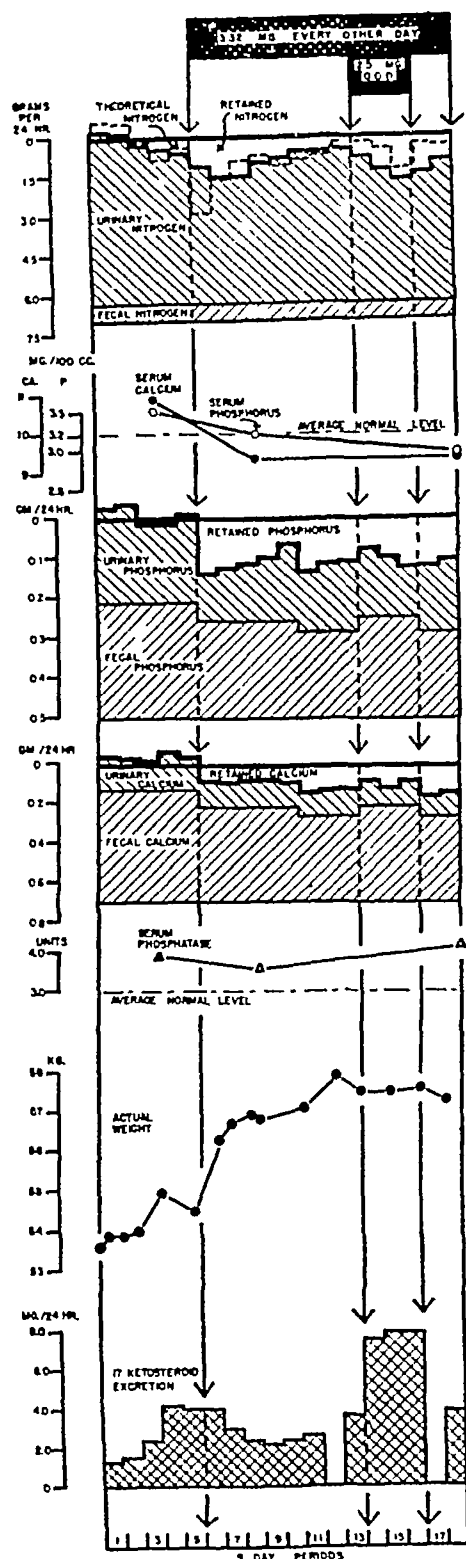


Fig. 6 Case 2 (E. P , M G H 203540) Effect of Estradiol Benzoate and Testosterone Propionate on Nitrogen, Phosphorus, and Calcium Balances, on Serum Calcium, Phosphorus, and Alkaline Phosphatase Levels, on Body Weight and on Urinary 17-Ketosteroid Excretion

when lifting. Physical examination showed a nervous woman with a tremor of her head, and considerable deformity of her back. Her blood pressure was 115/75. X-ray examination revealed extensive osteoporosis with multiple fractured vertebrae; bones of skull were approximately normal in density. Laboratory studies no abnormalities of the urine, stools, or blood cells, urine calcium 2 to 4 plus by the Sulkowitch test, serum calcium 10.6 mgm. per cent, serum phosphorus 3.1 mgm. per cent, serum alkaline phosphatase 3.7 Bodansky units, serum chloride 93.2 m.eq. per l., non-protein nitrogen level 26 mgm. per cent; and total protein 7.8 grams per cent with an albumin/globulin ratio of 1.7. Electrocardiographic tracing was normal, follicle-stimulating hormone excretion in the urine was high (consistent with the menopause).

The metabolic data of Case 3 are given in Fig. 7. The study, conducted in 6-day periods, consisted of· 1) four control periods; 2) four periods on methyl testosterone, 40 mgm. by mouth daily, 3) five periods in which 1.66 mgm. of estradiol benzoate daily by injection were added to the methyl testosterone therapy, 4) five periods back on the methyl testosterone therapy alone; 5) four more control periods off medication; 6) three periods on pregnenolone, 30 mgm. intramuscularly daily, 7) four more control periods off medication; 8) five periods back on methyl testosterone, 40 mgm. by mouth daily with a change in the nitrogen and phosphorus intakes during the last 3 of these, and 9) one final period where the methyl testosterone therapy was increased to 100 mgm. by mouth daily. The urinary determinations were made on 3-day periods throughout.

In Fig. 7 it should be noted first that the theoretical nitrogen balance is consistently less than the actual nitrogen balance, which indicates that there is some constant error throughout. Part of the error may be in the fecal nitrogen excretion which was not analyzed, but taken as 10 per cent of the nitrogen intake. In the absence of analyzed values, it would have been preferable, and the discrep-

For discussion, see text
The fecal nitrogen was estimated as 10 per cent of the intake The fecal phosphorus and calcium values as charted are averages of 2, 3, 4, or 5 five-day periods as follows· 1 through 5, 6 through 9, 10 through 13, 14 through 16, 17 through 18

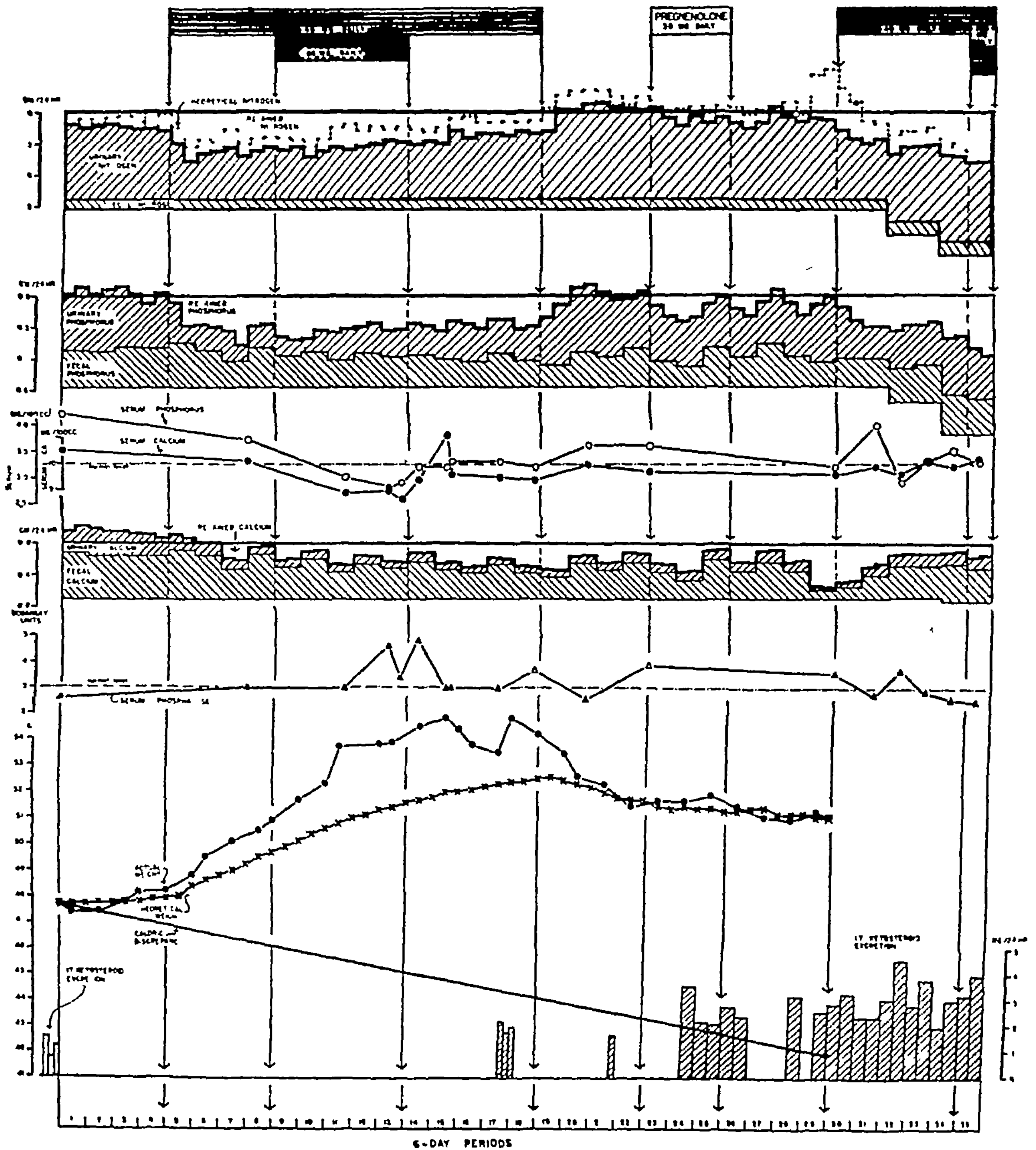


Fig. 7. Case 3 (R. W., M. G H 319940) Effect of Methyl Testosterone Alone and in Combination with Estradiol Benzoate, and of Pregnenolone on Nitrogen, Phosphorus, and Calcium Balances, on Serum Calcium, Phosphorus, and Alkaline Phosphatase Levels, on Body Weight, and on Urinary 17-Ketosteroid Excretion in a Female Patient with Post-Menopausal Osteoporosis

For discussion, see text

ancy would have been cut down, had we used the value of 1.283 grams per 24 hours, the average fecal nitrogen value for adults regardless of intake. The major part of the discrepancy is probably to be attributed to errors in the intakes. The daily diet was analyzed twice with the following results analysis October 1941 calcium 71 mgm., phosphorus 584 mgm.,

and nitrogen 9.31 grams; analysis February 2, 1944: calcium 64 mgm., phosphorus 611 mgm., and nitrogen 8.40 grams. Fig. 7 was constructed from the analysis of 1941; had it been constructed from the analysis of 1944, the discrepancy would have been almost eliminated. Thus if one recalculates on the basis of the 1944 analysis, the theoretical nitrogen balance of period 4b, and in addition uses the value of 1.283 grams for the fecal nitrogen instead of 10 per cent of the intake, one obtains the values +0.65 and +0.45 grams for the theoretical and actual nitrogen balances, respectively, in contrast to the values of +0.18 and +1.71 grams. Sir' e the above discrepancy is fairly constant, it does not affect the trends induced by treatment.

Fig. 7 is self-explanatory. To be noted are: 1) the decrease in the nitrogen, phosphorus, and calcium excretions with methyl testosterone therapy, and the rebound of nitrogen and phosphorus excretions on cessation of therapy; 2) the fact that the fecal, as well as the urinary, excretions of both calcium and phosphorus were reduced under methyl testosterone therapy, 3) the fact that there was not an immediate rebound of the calcium excretion following cessation of methyl testosterone therapy; 4) the futher improvement in the calcium balance, but not in the nitrogen balance, when estradiol benzoate therapy was added to the methyl testosterone therapy (periods 9 to 13); 5) the fall in serum phosphorus level with methyl testosterone and especially with estradiol benzoate therapy; 6) the definite tendency of the serum calcium level to parallel the serum phosphorus level (see also Fig. 6); and 7) the failure of the serum phosphatase level to show a significant change. The effect of the pregnenolone therapy is inconclusive; it did not significantly affect the very low 17-ketosteroid excretion. No explanation is forthcoming in periods 29 and 30 for the low fecal calcium excretions not associated with low nitrogen and phosphorus excretions; as a result, the data during periods 30 through 36 are difficult to interpret. The actual and theoretical weight curves suggest that there was retention of extracellular fluid with methyl testosterone therapy, which was augumented when estradiol benzoate therapy was added. Pregnenolone therapy had a minimal effect on extracellular fluid retention.

Case 4. Senile Osteoporosis in a Male of 72 Testosterone Propionate and Estradiol Benzoate Therapy.

M. H. (M. G. H. 278511), a male of 72 years, developed pain in the back after a minor injury one year before admission (1-1-41). The symptoms persisted in spite of local therapy, and he was referred to the hospital. The only abnormal findings on physical examination were a thin skin and deformities of the spine; his blood pressure was 140/80. X-ray examination of the spine showed marked decrease in density of the vertebrae with a codfish deformity of some, and wedging or collapse of others. Laboratory studies: serum calcium 10.0 mgm. per cent; serum phosphorus 3.1 mgm. per cent; serum alkaline phosphatase 4.2 Bodansky units, serum total protein 7.0 grams per cent; nonprotein nitrogen 18 mgm. per cent; urinary 17-ketosteroid excretion 7.2 and 6.9 mgm. per 24 hours, follicle-stimulating hormone excretion in the urine normal, gastric acidity normal. The normal level of the follicle-stimulating hormone excretion is evidence against the idea of the osteoporosis having been due to the "male menopause."

The metabolic data of Case 4, which comprise studies done on 290 of 530 consecutive days, are shown in Fig. 8. The study, conducted in 5-day periods, consisted of: 1) five control periods; 2) five periods on testosterone propionate, 25 mgm. by injection daily; 3) five periods in which estradiol benzoate 1.66 mgm. by injection on alternate days was added to the testosterone propionate therapy; 4) five periods back on testosterone propionate alone; 5) seven control periods off all medication; 6) five periods on estradiol benzoate 1.66 mgm. by injection twice daily; 7) ten days without collections on the same medication; 8) two more periods on the same medication; 9) ninety-three days at home on estradiol benzoate 3.32 mgm. by injection 3 times a week; 10) five periods on the same therapy; 11) nine periods in which testosterone propionate 25 mgm. intramuscularly daily was added to the estradiol benzoate therapy, during the last three of which periods the intakes of nitrogen and phosphorus were markedly increased; 12) three periods on the same diet and the same estradiol benzoate therapy but off testosterone pro-

pionate therapy, 13) ninety-one days at home on the same estradiol benzoate therapy, 14) three periods on the original diet without change in the estradiol therapy, 15) forty-three days at home off all medication, and finally 16) four control periods on the original diet without medication.

Fig. 8 is self-explanatory. The observations as a whole confirm those noted in Cases 1 to 3 with postmenopausal osteoporosis.

Again, as in Case 3, the theoretical nitrogen balance as charted is consistently less positive than the actual nitrogen balance which suggests some constant error. This discrepancy is probably to be attributed to errors in the intakes and to estimation of the fecal nitrogen as 10 per cent of the nitrogen intake (see discussion under Case 3). Case 4 received the same diet as Case 3, this diet was analyzed twice with the results given in the discussion under Case 3. Fig. 8 was constructed from the analysis of 1941, had it been constructed from the analysis of 1944, the discrepancy would have been almost eliminated. Thus, if one recalculates on the basis of the 1944 analysis, the theoretical nitrogen balance of period 5, and in addition uses the value of 1.283 grams for the fecal nitrogen instead of 10 per cent of the intake, one obtains the values +0.87 and +1.30 grams for the theoretical and actual nitrogen balances, respectively, in contrast to the values of +0.41 and +2.21 grams. As was pointed out in connection with Case 4, since the above discrepancy is fairly constant, it does not affect the trends induced by treatment.

To be noted especially in Fig. 8 are 1) the marked reduction in nitrogen, phosphorus, and calcium excretions with testosterone therapy; 2) the lack of rebound in the calcium excretion as opposed to nitrogen and phosphorus following cessation of testosterone therapy, 3) the further reduction in the phosphorus and especially in the calcium excretion, but not in the nitrogen excretion, when estradiol benzoate therapy was added to testosterone propionate therapy (periods 16 to 20), 4) the improvement in all three balances when testosterone propionate was added to estradiol benzoate therapy (periods 40 to 45), 5) reduction in the fecal as well as the urinary calcium and phosphorus excretions by both testosterone propionate and estradiol benzoate therapy, 6) the effect of both testosterone propionate and estradiol benzoate therapy in lowering the serum phosphorus level, 7) the failure of marked increases in the nitrogen and phosphorus balances by increased diet to affect the calcium balance (periods 46, 47, 48), 8) the absence of any significant change in the serum phosphatase and calcium levels, 9) the fall in the urinary 17-ketosteroid level with estradiol benzoate therapy, and 10) the tendency to accumulate extracellular fluid during both testosterone propionate and estradiol benzoate therapy as suggested by the theoretical weight curves, with a prompt loss following the cessation of therapy.

A more detailed analysis to emphasize the close agreement between the nitrogen, phosphorus, and calcium balances of periods 1 through 22 is shown in Fig. 2. It will be seen that measured phosphorus balance very nearly can be accounted for by the sum of the theoretical phosphorus balances based on the measured calcium and nitrogen balances during both the control observations and the experimental periods. This analysis supports the contention that nearly all of the phosphorus retained as a result of testosterone propionate therapy is retained either as bone or as protoplasm.

Case 5 "Normal" Female, Effect of Orthopedic Operation, No Specific Therapy.

E. S. (M. G. H. 360207), a female of 35 years, had poliomyelitis at the age of nine, involving the left leg alone, and since the age of fourteen had worn a 6-pound brace on the left leg. She had always been very active. For the ten years prior to study she had had metatarsal pain in the right foot, and for three years had turned her right ankle frequently. She was admitted for a triple arthrodesis and muscle transplant to strengthen the right ankle. The menstrual history was normal. From the point of view of the experiment the patient can be considered a normal adult female in every respect, except for the residuals of the poliomyelitis of the left leg, her blood pressure was 120/80. Laboratory studies serum calcium 9.8 mgm. per cent, serum phosphorus 3.5 mgm. per cent, serum alkaline phosphatase 2.4 Bodansky units, and serum total protein 4.7 grams per cent, urinary 17-ketosteroid excretion 7.6 mgm. per 24 hours.

The metabolic data of Case 5 are shown in Fig. 9. Throughout the entire experiment the patient was on a constant, neutral-ash, low calcium diet, except for the immediate post-

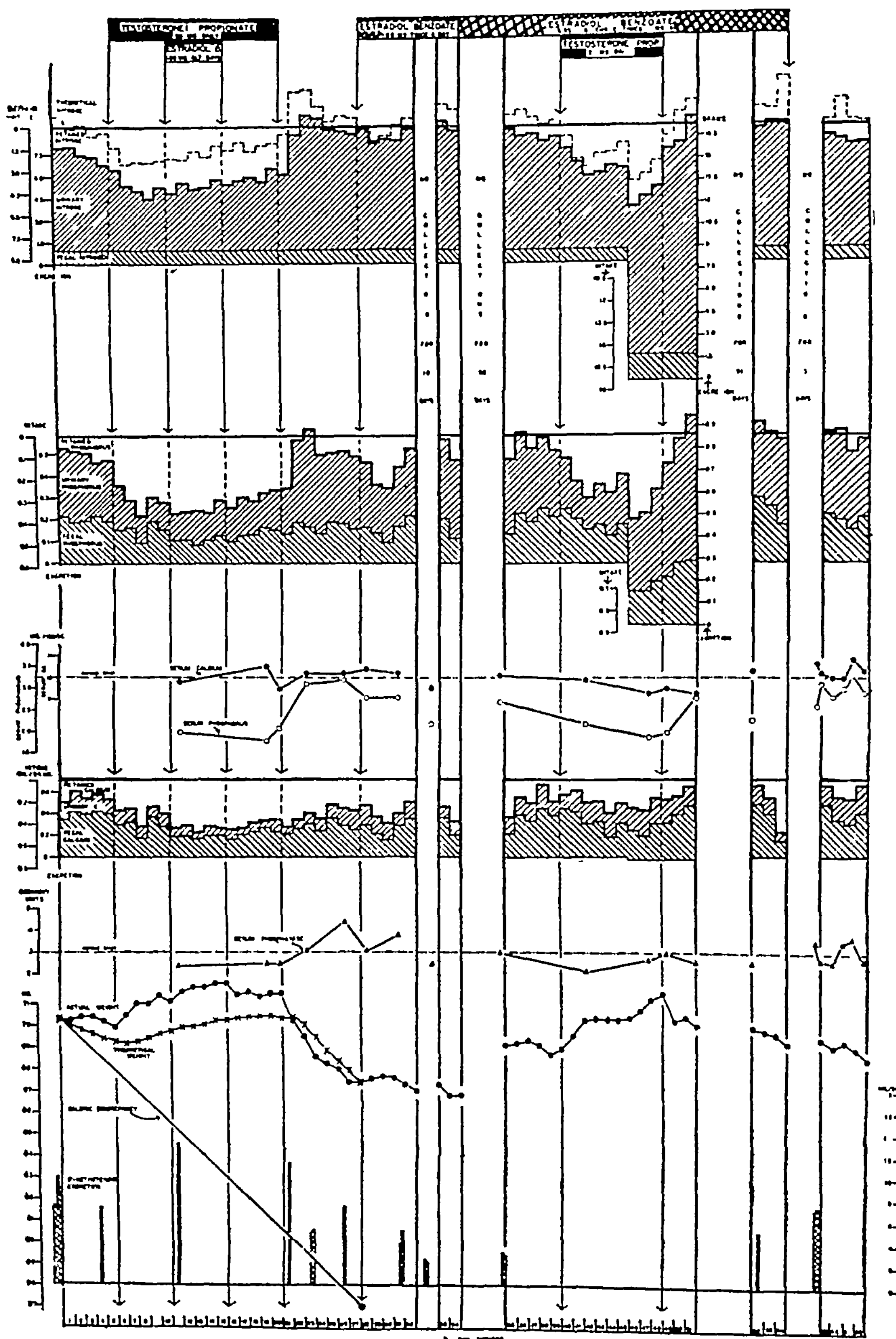


Fig. 8. Case 4 (M H , M. G H 278511) Effect of Testosterone Propionate Alone and in Combination with Estradiol Benzoate and vice versa on Nitrogen, Phosphorus, and Calcium Balances, on Serum Calcium, Phosphorus, and Alkaline Phosphatase Levels, on Body Weight, and on Urinary 17-Ketosteroid Excretion in a Male Patient with Senile Osteoporosis

For discussion, see text

operative period. She was up and active during the preoperative period, and immobilized in a cast from the foot to the hip after operation. She underwent an arthrodesis of the right foot on the second day of period 5, there were no analyses for metabolic data during periods 5 and 6, but the 17-ketosteroid excretion was followed.

During the four control periods the patient was in negative calcium and phosphorus balance, the former was of the order of magnitude one would expect with patients on this diet. As expected, there was a marked increase in the calcium excretion after the operation, which persisted unabated to the end of the investigation (58 days after the operation). The increase in calcium excretion was not entirely in the urine. The 17-ketosteroid excretion was normal preoperatively, which confirms the contention that she was not debilitated; it rose immediately after operation, and then fell decidedly below the preoperative level for about 20 days. The pattern of response was thus similar to that encountered following any traumatizing event. The marked elevation in 17-ketosteroid excretion in period 11 coincided with the patient being allowed up in a wheel chair.

Periods 7 through 14 in this untreated case serve as a control for similar studies in Cases 8 and 9, who received estradiol therapy during the postoperative period (Fig. 12).

Case 6· Multiple Traumatic Fractures with Operative Reduction of One in a Previously "Normal" Male, Effect of Estradiol Benzoate Therapy.

H. D. (M. G. H. 382395), a fireman of 50 years, fell three stories and suffered fractures of ribs, pelvis, right tibia and right fibula, and multiple contusions and abrasions. The patient was in shock on admission, but responded promptly to a blood transfusion. On physical examination he was found to be a well-preserved man without organic disease, blood pressure was 110/60. A Kirschner wire was inserted through the os calcis and a Zimmer bow applied. During the next two weeks the fractures were reduced by traction and by several manipulations under anesthesia. The patient was transferred to the metabolic ward where studies were begun 44 days after the accident. Laboratory studies· serum calcium

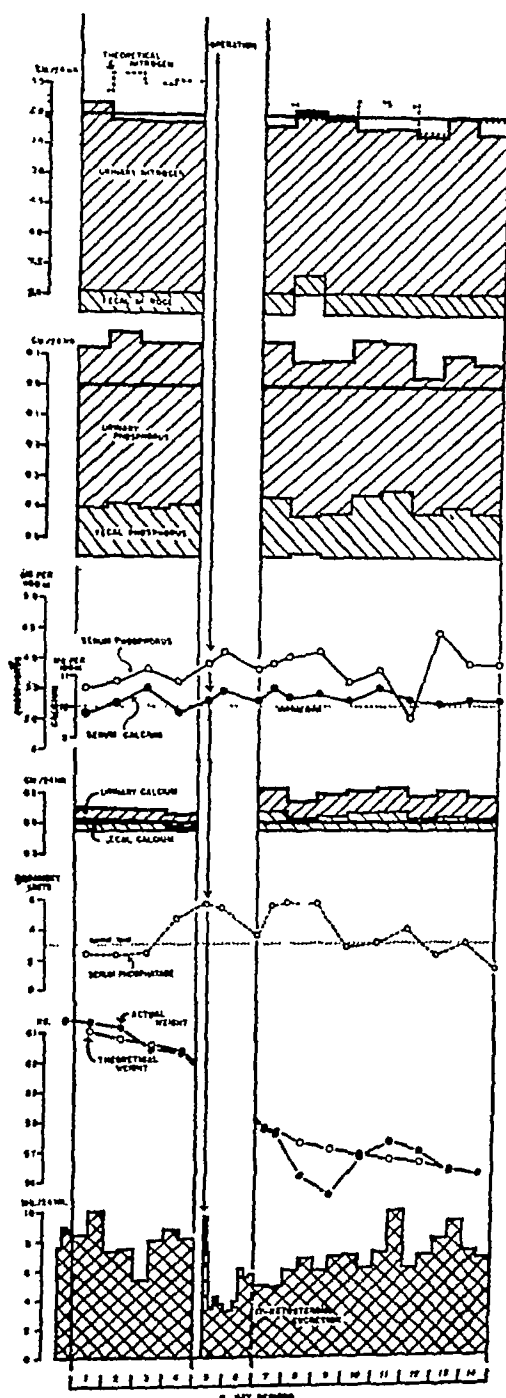


Fig 9 Case 5 (E S., M G. H 360207) *Nitrogen, Phosphorus, and Calcium Balances, Serum Calcium, Phosphorus, and Alkaline Phosphatase Levels, Weight, and Urinary 17-Ketosteroid Excretion in a Female Patient with Osteoporotic Process Induced by Operation and Immobilization*

For discussion, see text

10.7 mgm. per cent, serum phosphorus 3.3 mgm. per cent, serum alkaline phosphatase 2.7 Bodansky units, serum total protein 6.7 grams per cent.

The metabolic data of Case 6 are shown in Fig. 10. The study, conducted in 3-day periods, consisted of 1) six control periods, 2) six periods in which 1.66 mgm. of estradiol benzoate was given daily by injection; and 3) six-post-treatment control periods. The stool periods were analyzed two at a time.

Fig. 10 is self-explanatory. The most important observations concern the calcium metabolism, these are better shown in Fig. 12 and will be discussed below. Again, there was a fall in the serum phosphorus, and, if anything, a fall in the serum phosphatase. Of interest is the fall in 17-ketosteroids in period 13, followed by the rise in urinary nitrogen, phosphorus, and calcium in period 14. We believe these to be connected though unexplained phenomena.

Case 7 Bone Grafting Operation in an Ununited Femur of an Otherwise "Normal" Male, Effect of Estradiol Benzoate Therapy.

C.M. (M.G.H. 348774), a male of 24 years, sustained a fracture of the pelvis and of the right femur in an automobile accident nine months before study. The femur failed to unite properly and, although the patient was active and able to walk about with a cane, he had unusual motion and instability in his right femur because of the poor union. He was readmitted for bone grafting. Physical examination revealed a young adult male who was normal in all respects except for the incomplete union of his right femur, his blood pressure was 105/60. Laboratory studies serum calcium 10.3 mgm. per cent, serum phosphorus 4.5 mgm. per cent, serum alkaline phosphatase 2.9 Bodansky units, and serum total protein 6.0 grams per cent.

The metabolic data of Case 7 are shown in Fig. 11. The study, conducted in 3-day periods, consisted of 1) six control periods, 2) six periods in which 1.66 mgm. of estradiol benzoate was given daily by injection, and 3) six-post-treatment control periods. The stool periods were analyzed two at a time.

Fig. 11 is self-explanatory. The theoretical nitrogen balance shows a constant deviation

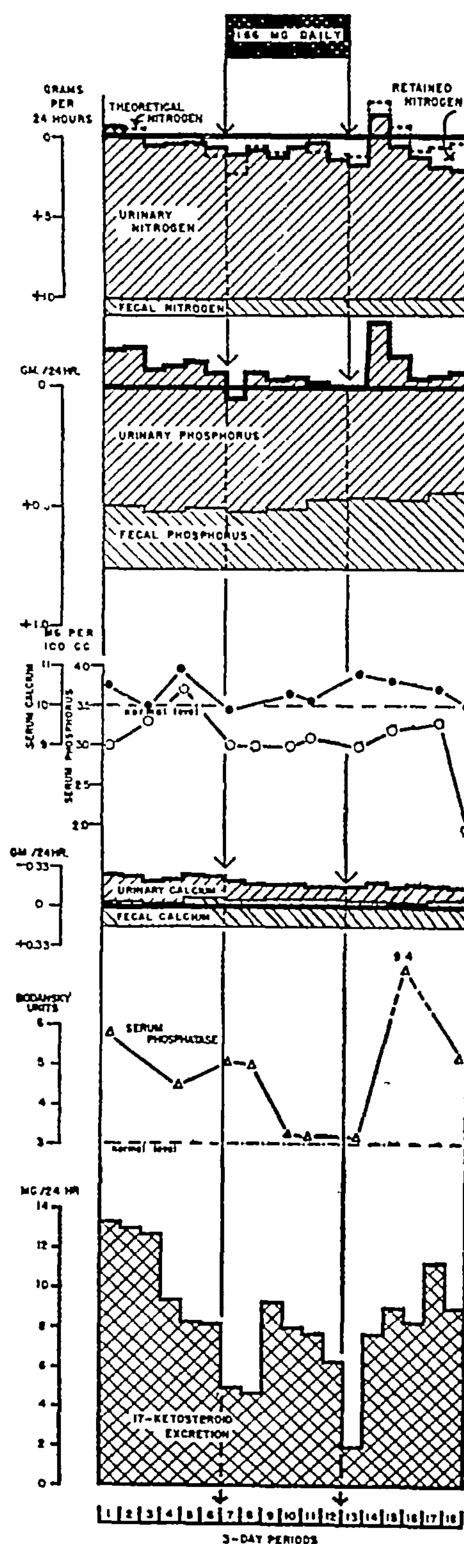


Fig 10 Case 6 (H. D., M G H. 382395) Effect of Estradiol Benzoate on Nitrogen, Phosphorus, and Calcium Balances, on Serum Calcium, Phosphorus, and Alkaline Phosphatase Levels; and on Urinary 17-Ketosteroid Excretion in a Male Patient with Osteoporotic Process Induced by Multiple Fractures, Operation, and Immobilization

For discussion, see text

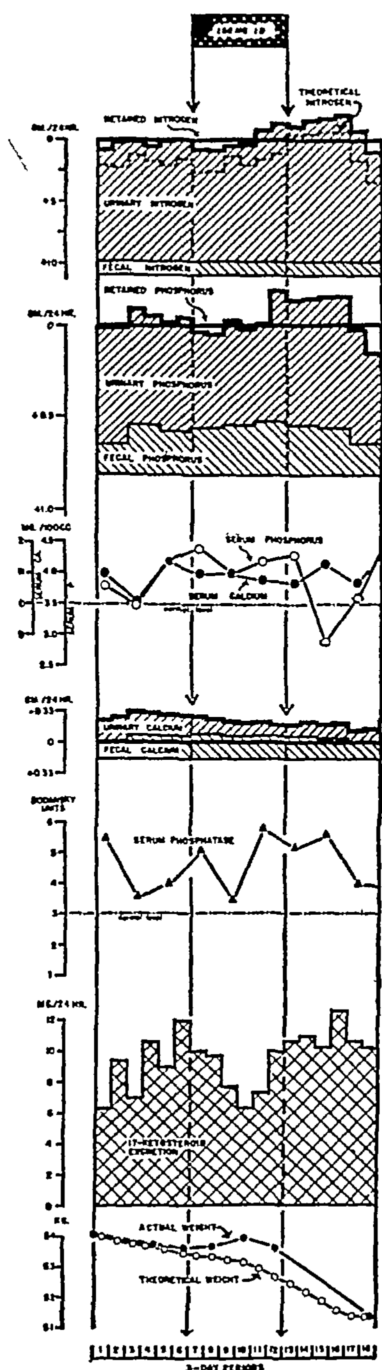


Fig 11 Case 7 (C. M , M G. H 348774) Effect of Estradiol Benzoate on Nitrogen, Phosphorus, and Calcium Balances, on Serum Calcium, Phosphorus, and Alkaline Phosphatase Levels, on Urinary 17-Ketosteroid Excretion and on Weight in a Male Patient with Osteoporotic Process Induced by Operation and Immobilization.

For discussion, see text

from the measured nitrogen balance, which suggests some constant error (see above). The calcium data, as in Case 6, are better shown in Fig. 12, and will be discussed below. It should be noted that the serum phosphorus in

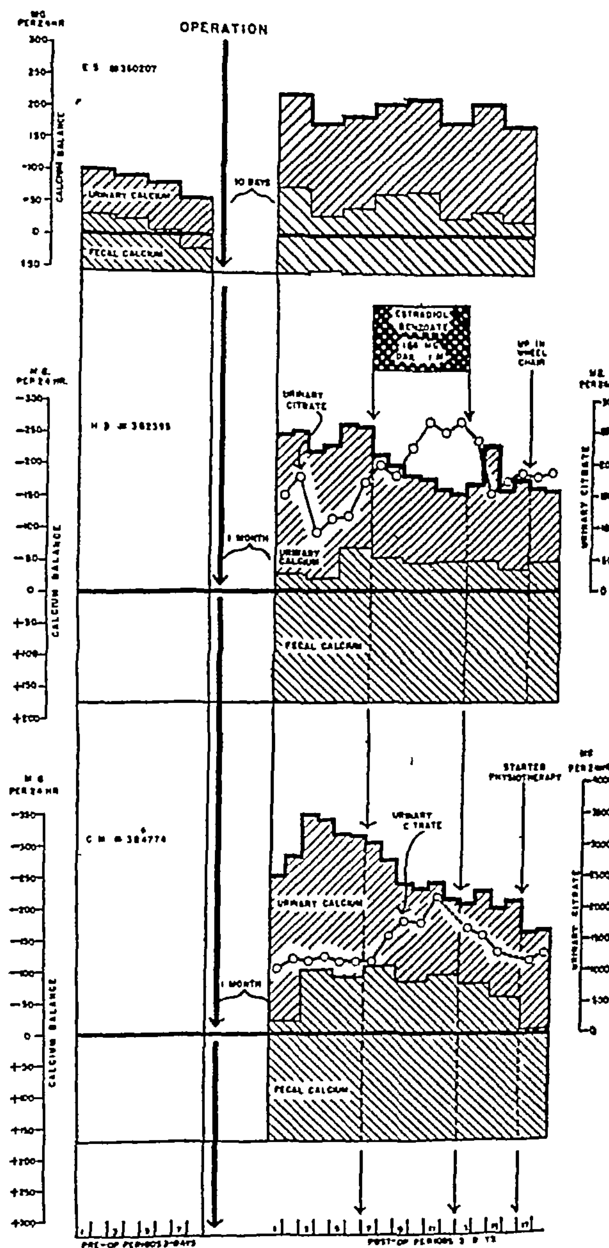


Fig 12 Metabolic Data for Calcium of Cases 5, 6, and 7 Effect of Estradiol Benzoate as Compared with No Therapy on the Calcium Balances in Patients with Osteoporotic Process due to Operation and Immobilization.

For discussion, see text

this case, as opposed to all the other cases, did not fall during estradiol therapy. The 17-ketosteroid excretion showed a tendency to fall during the estradiol benzoate therapy, which is also somewhat suggested in Fig. 10.

In Fig. 12 the calcium data of Cases 6 and 7 with estradiol benzoate therapy are compared with those of Case 5 without such therapy. It is quite clear that estradiol benzoate therapy resulted in a decrease in the urinary calcium excretion, but had little effect on the fecal calcium excretion during the 18 days of administration. However, the tendency for the fecal calcium to decrease in Case 7 after the therapy was stopped may well have been delayed response to the therapy. The urinary citric acid values carried out and interpreted by Dr. Ephraim Shorr confirm his finding of a rise during estrogen therapy.

Case 8: Cushing's Syndrome with Osteoporosis; Nephrolithiasis; Estradiol Benzoate and Testosterone Propionate Therapy.

B. V. (M. G. H. 74372) a 25-year-old, single girl of Portuguese descent, has been followed at the Massachusetts General Hospital for five years with the diagnosis of Cushing's syndrome. Her chief complaint when first seen was, "amenorrhea for two years."

She also revealed that she had been gaining weight about the face, neck, and abdomen, and that her face had become ruddy and more hairy than before. In addition, she had noticed easy bruisability and had had recurrent skin infections.

On examination, she had the typical round, full, plethoric facies of Cushing's syndrome, with excessive facial and abdominal hair. The skin appeared atrophic with purplish striae and numerous visible dilated subcutaneous blood vessels. There were scars of old infections on the legs. The optic fundi showed some old hemorrhage and exudate. The heart was not enlarged. Blood pressure varied from 130/80 to 170/110. The clitoris was not enlarged.

Laboratory studies included· negative Hinton test result, R. B. C. 4.8; Hgb. 95% (T); urine, a trace of albumen, occasional glycosuria, *B. coli* and *Staphylococcus albus* on culture; blood chemistry, not remarkable, sodium normal. Glucose tolerance test showed a failure to return to normal four hours after administration of glucose. Combined glucose and insulin test result paralleled that of the glucose tolerance test. Several B.M.R.'s were normal. Electrocardiogram showed evidence of myocardial, damage. Follicle stimulating hormone assay on urine was negative for ten mouse units. The 17-ketosteroids in the urine varied from 8 to 14 mg./24 hours before treatment. X-rays showed marked osteoporosis with numerous fractured vertebrae and bilateral kidney stones.

Both adrenals were explored. Biopsied sections were said to show no abnormalities. She received three courses of x-ray radiation to the pituitary. There was a remission for about a year, with return of regular menses after the first course. The last two courses had no effect. Kidney stones required operation twice.

She showed marked improvement on testosterone therapy, as discussed below. It is of real interest that after cessation of therapy, her improvement continued and it was found that the abnormality in sugar metabolism had entirely disappeared. For two years after cessation of therapy, her menses were normal and she presented no evidence of a recurrence. Subsequently she exhibited increasingly severe cardiac disease, from which she ultimately died.

The metabolic data of Case 8 are shown in Fig. 13. The study covers 37 five-day periods obtained on four hospital admissions. The data in Fig. 13 are self-explanatory. It should first be noted that the phosphorus balance corresponds reasonably well with the sum of the nitrogen and calcium balances during the last 23 periods but not the first 14. This suggests some constant error in the first 14 periods, probably the value for the nitrogen intake. A more detailed analysis to emphasize the close agreement between the nitrogen, potassium, phosphorus, and sulphur balances of periods 15 through 33 is shown in Fig 3. It will be seen that there is a close correspondence between the measured and the theoretical nitrogen balances based on potassium, phosphorus, and sulphur. This is evidence that testosterone propionate therapy induced a retention of nitrogen, potassium, phosphorus, and sulphur in the proportions that exist in muscle protoplasm.

Although it was originally concluded from

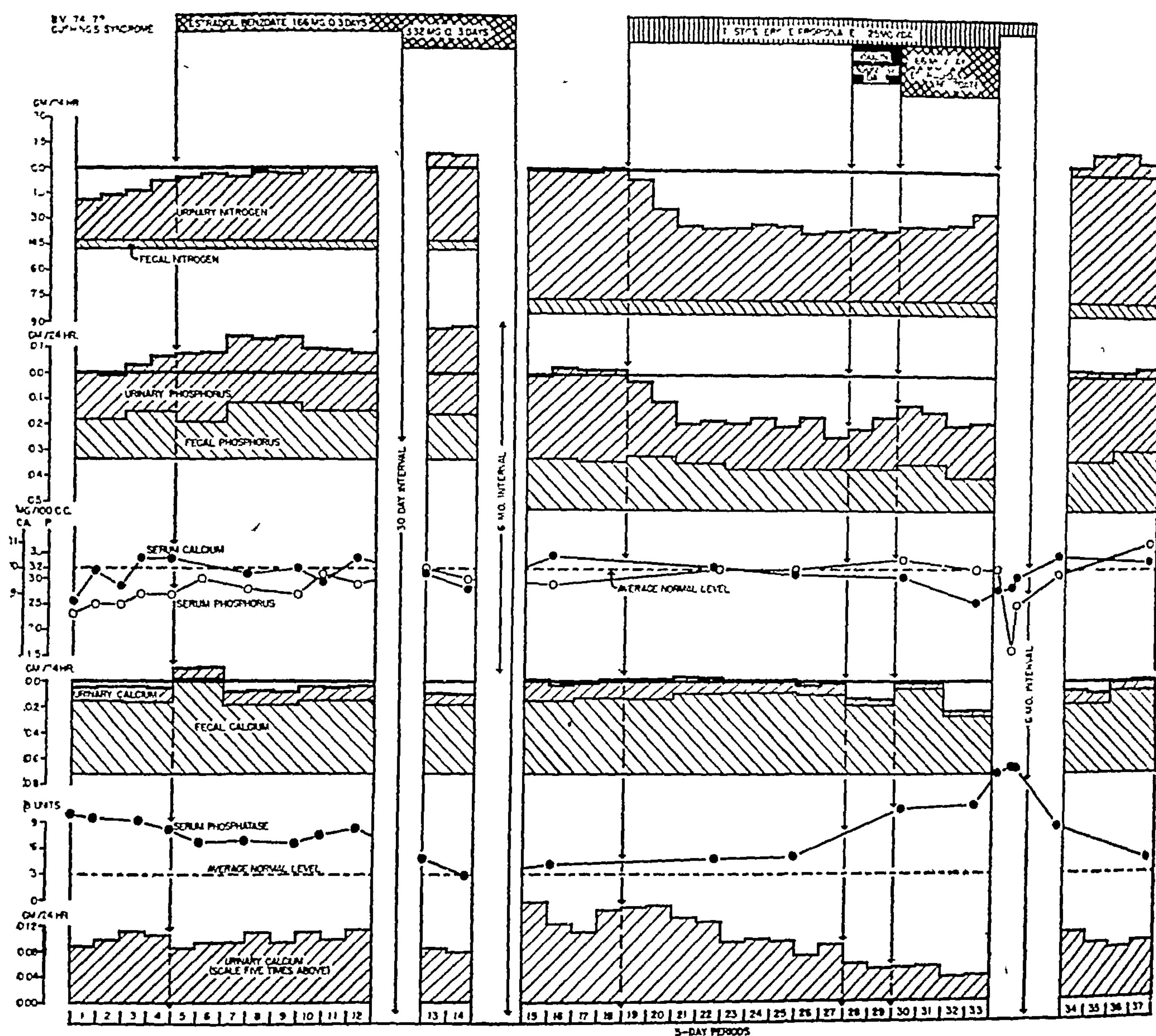


Fig. 13 Case 8 (B. V., M. G. H. 74372) Effect of Estradiol Benzoate and Testosterone Propionate on Nitrogen, Phosphorus, and Calcium Balances, and on Serum Calcium, Phosphorus and Alkaline Phosphatase in a Female Patient with Osteoporosis due to Cushing's Syndrome.

For discussion, see text
At the bottom of the chart, the urinary calcium is shown separately on an enlarged scale

these studies that estrogen was without beneficial effect, this was true with respect to the nitrogen balance but not altogether true with respect to the calcium balance Thus, with the larger dose of estradiol benzoate in periods 13 and 14 there is an increase, probably significant, in the calcium balance. Furthermore, when estradiol benzoate was added to testosterone propionate therapy in periods 30 through 33, there was a further fall in the urinary calcium excretion and an increase in the positive calcium balance Other observations to be underlined in Fig. 13 are: 1) the marked decrease in the urinary nitrogen, phosphorus, and calcium excretions with testosterone propionate therapy, 2) the marked rise in the serum phosphatase level when the increase in calcium balance became appreciable (see pe-

riods 30 through 33). Whereas Fig. 13 suggested that insulin had a marked effect on calcium balance (see periods 28 and 29) we are inclined to discount this because of the essentially negative result in a second patient with Cushing's syndrome so treated

Case 9 Cushing's Syndrome with Osteoporosis in a 13-year-old Girl; Testosterone Propionate Therapy, X-ray Irradiation of the Pituitary

I. G (M. G. H. 350260) a 13-year-old girl had Cushing's syndrome for over 5 years. Although metabolic studies have been done on this girl, they will not be reported at this time Instead, I will present some very interesting x-ray changes which illustrate the predilection of "S" hormone of the adrenal cortex for the spine and pelvis.

Whereas it is a general rule that a hormone, if it exerts an effect at all on a tissue, does so throughout the entire tissue, this does not mean that there may not be sites of predilection In the case of the "S" hormone, as judged by its effect on patients with Cushing's syndrome, there is a marked predilection for the spine and pelvis. The skull is much less involved and the extremities almost not at all Conversely, if one were to recover from Cushing's syndrome, one would expect to see changes in the spine and pelvis and no changes in the extremities

In Figs. 14-17 are shown the x-ray findings of the lumbar and lower thoracic spine of a female patient (I. G , 350260, of 15 years at time of second x-ray (7-10-45)) before (Figs. 14 and 16) and approximately fifteen months after (Figs. 15 and 17) cessation of the activity of her Cushing's syndrome. A more detailed case history will appear elsewhere, suffice it is to say that the agent responsible for overcoming the activity is not definitely known The improvement may have been spontaneous, it may have been the result of testosterone therapy, it may have been the result of x-ray irradiation of the pituitary. The time sequence suggests the last It will be noted in Fig. 17 that the intervertebral discs were squeezed together again as a result of the growth pressure. In Fig. 18 are depicted, in diagrammatic form, the author's interpretation of· A) the normal vertebral findings in an individual her age, B) the findings in the patient before recovery, and C) the findings in the patient after recovery



Fig 14 Photograph of an X-ray of the Pelvis and Lumbar Spine of a Girl (I.G) with Cushing's Syndrome Before Recovery

Film taken June 6, 1942

Fig. 19 represents a tracing of the vertebrae shown in Figs. 16 and 17, together with an analysis of the comparative widths of the vertebrae and intervertebral discs before and after recovery.

Note in Figs. 15 and 17 that the marked change in the vertebrae following recovery is almost entirely due to the laying down of dense new bone, that bone which existed before recovery remains very osteoporotic It was only because this patient was still growing that these remarkable changes could be demonstrated It takes a long time in the adult to demonstrate increased density of the vertebrae after alleviation of the activity in Cushing's syndrome. In Fig. 20 is seen the x-ray of the wrist taken approximately fifteen months after recovery One is impressed by the lack of a zone of increased density adjacent to the



Fig 15 Photograph of an X-ray of the Pelvis and Lumbar Spine of a Girl (I G) Approximately Fifteen Months After Recovery from Cushing's Syndrome (cf. Figure 14)

Film taken July 3, 1945 Note that intervertebral discs have been squeezed together, and that centers of vertebrae remain radio-translucent in marked contrast to dense bone which has been newly laid down For further analysis of changes see Figures 18 and 19



Fig 16 Photograph of an X-ray of the Lumbar Spine of a Girl (I G.) with Cushing's Syndrome Before Recovery

Film taken June 6, 1942

radial epiphyseal cartilage, this is strong evidence that the "S" hormone was not affecting the bone being laid down at the wrist

The remarkable physical changes in this girl are shown in Figs. 21 and 22

Conclusions from Metabolic Studies

1. Estrogens (estradiol benzoate and diethylstilbestrol) decreased the calcium and phosphorus excretions in the types of osteoporosis studied

Additional Observations

a The fecal as well as the urinary calcium and phosphorus excretion were decreased in most cases.

b The effects were manifested within 6 days, were maximal in 30 days, and persisted 30-50 days after drug was stopped.

c. The serum inorganic phosphorus level tended to be high in postmenopausal cases and to fall with estrogen

d The serum alkaline phosphatase level *did not* rise

e The urinary nitrogen excretion showed a poorly sustained decrease

f. There was no convincing evidence that large doses were better than small, except with stilbestrol (one case).



Fig 17 Photograph of an X-ray of the Lumbar Spine of a Girl (I G) Approximately Fifteen Months After Recovery from Cushing's Syndrome (cf Figure 16)

Film taken July 3, 1945 Note that intervertebral discs have been squeezed together, and that centers of vertebrae remain radio-translucent in marked contrast to dense bone which has been newly laid down For further analyses of changes see Figures 18 and 19

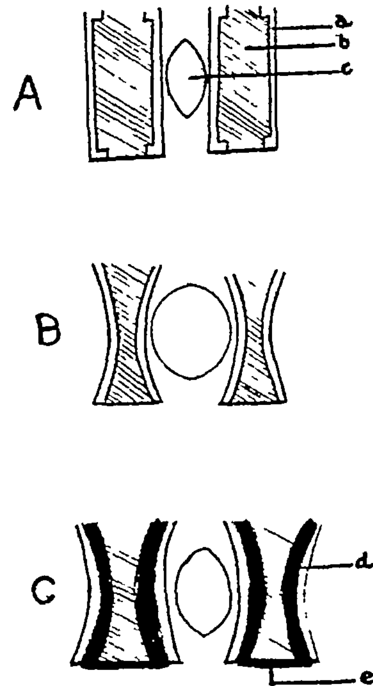


Fig 18 Schematic Representations of Interpretation of Vertebral Changes in Figures 14 and 16 Before Recovery (B), in Figures 15 and 17 Approximately Fifteen Months After Recovery (C), as Compared with Normal Findings for the Same Age (A)

a = cartilaginous end-plate of vertebra; b = bony part of vertebra, c = nucleus pulposus, d = bone laid down after recovery by cartilaginous end-plate and e = bone laid down after recovery by periosteum Note in (B) that nucleus pulposus is expanded at expense of vertebrae because of decreased resistance of vertebrae Note in (C) that nucleus pulposus has been partially squeezed together again by growth pressure

2. Androgens (testosterone propionate and methyl testosterone) like estrogens decreased the calcium and phosphorus excretions in the types of osteoporosis studied.

Additional Observations

Like Estrogens

a. The fecal as well as the urinary calcium and phosphorus excretions were decreased.
b. The effects on calcium were slow in reaching a maximum, and persisted for a long time after drug was stopped.
c. The serum inorganic phosphorus level tended to fall with androgen.
d. The serum alkaline phosphatase level *did not* rise except in the case of Cushing's syndrome.

Unlike Estrogens

e The urinary nitrogen excretion decrease was marked and prolonged
f. Methyl testosterone therapy was as effective as testosterone propionate therapy on the calcium metabolism.

3. Progesterone therapy had no definite effect alone or with estrogens.
4 The combination of estrogen and androgen medication was better than either therapy alone.

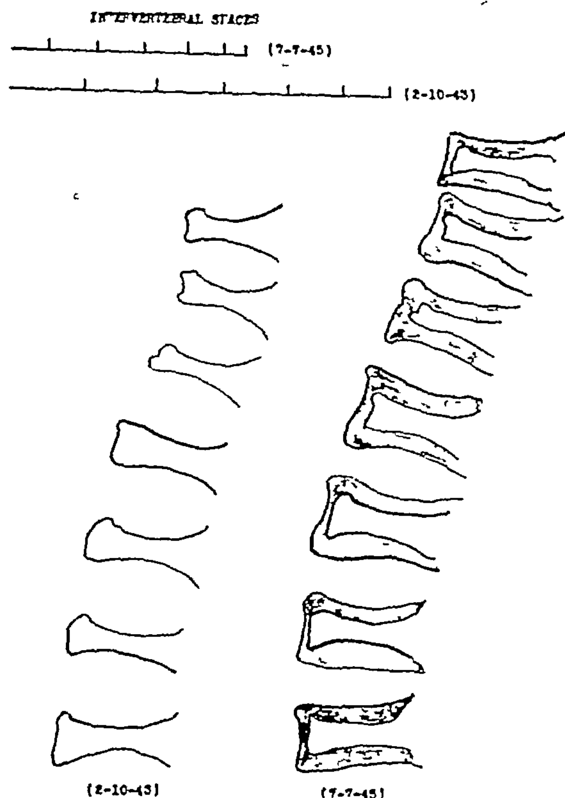


Fig 19 Tracing of Vertebrae Shown in Figures 16 and 17 (A) Vertebrae on 2-10-43 Before Recovery, (B) Vertebrae on 7-10-45 Approximately Fifteen Months After Recovery

Note that osteoporotic centers of vertebrae in (B) have same configuration as whole vertebrae on 2-10-43 (A) Note that the sum of the widths of the six intervertebral discs in (B) is shorter than the sum in (A) in spite of the fact that the total length of the vertebrae in (B) is greater than the total length in (A)



Fig 20 Photograph of an X-ray of the Wrists of a Girl (I G) Approximately Fifteen Months After Recovery from Cushing's Syndrome

Film taken July 3, 1945 Note absence of any change in density of that bone most recently laid down in juxtaposition to the epiphyseal cartilages

Certain Therapeutic Aspects Concerning Postmenopausal Osteoporosis

During the past five years a large number of cases, many complicated by fractures, have been treated with estrogens alone and in combination with testosterone compounds As a group, these patients have responded very satisfactorily Within weeks to months, the pain in the spine and other bones usually has been considerably relieved or completely eliminated. There has frequently been an increase in weight, apparently an increase in the thickness of the skin and an improvement in the general well-being Whereas the study is impossible to control, we have the impression that fractures, especially of the hip, in old ladies have responded better than they would have otherwise However, in spite of these favorable clinical manifestations, it has been difficult to produce undisputed evidence that the bones (excluding fracture-sites) as visualized by x-ray have become more calcified than before the therapy was instituted. Nevertheless, recent films of several of the cases treated longest are fairly convincing

Dosages have ranged as follows diethylstilbestrol 0.5 to 1 mgm. daily p.o., estrone sulfate* 2 50 to 3 75 mgm daily p.o , estradiol benzoate 1.66 to 3 32 mgm 3 times a week i.m., and estradiol dipropionate 5 mgm weekly i m A few patients have been treated by implantation of pellets Excessive estrogenic effect on the endometrium has been controlled whenever a responsive uterus was present, by interrupting the estrogenic therapy periodically (every 4 to 6 weeks for 1 to 2 weeks), or by administering at regular intervals (every 4 to 6 weeks) a course of progesterone (5 mgm. daily i m for 5 days) or of anhydro-hydroxy-progesterone (40 to 60 mgm daily p.o for 5 days). Testosterone compounds cannot be giv-

*Conjugated equine estrogens [Premarin (Ayerst, McKenna, and Harrison)]

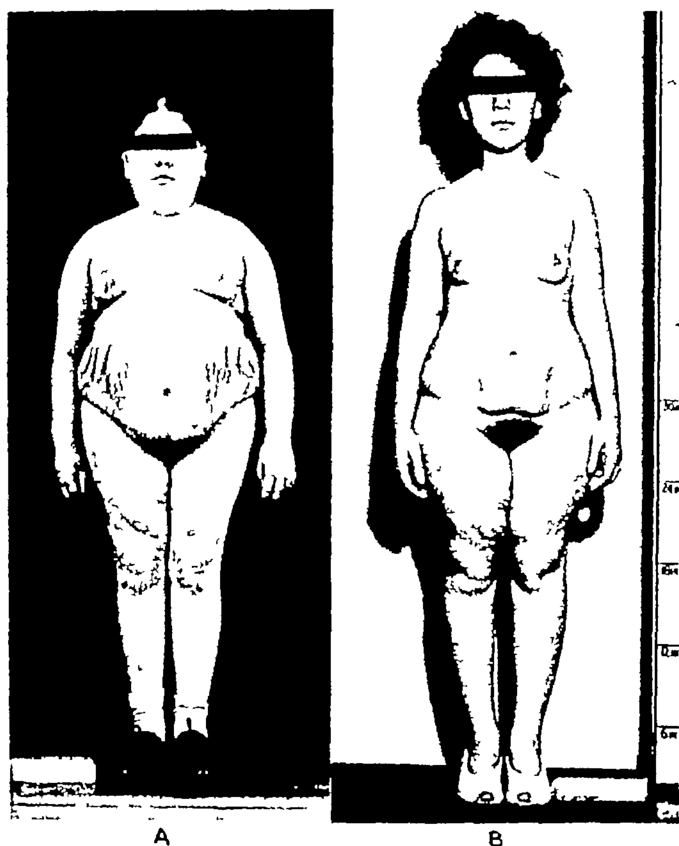


Fig. 21. Photographs to Contrast a Patient with Cushing's Syndrome Before and After Recovery

A = Patient I G (M G H 350620) in July, 1942 at 11 years and 10 months with Cushing's Syndrome before recovery, B = the same patient after recovery in July 1945 at 14 years and 10 months There is a separate scale for each photograph

en to most patients with the impunity suggested from Case 3, she was remarkably free from the masculinizing effect of such medication. Most women will not tolerate more than 300 mgm of androgen per month. We have given methyl testosterone 10 to 20 mgm. daily p o., and testosterone propionate 10 to 20 or 25 mgm. a week i m. One of the most successful methods of administering testosterone compounds to these patients is to implant one or two pellets of testosterone (75 mgm. each (Schering)) every 3 to 4 months. We usually give some form of testosterone at least for the first 6 to 12 weeks.

Since many of the steroids cause sodium retention, the above endocrine therapy may cause edema in certain elderly patients, especially if they have low serum protein levels If this is not controlled by a low sodium chlo-



Fig. 22. Photographs to Contrast a Patient with Cushing's Syndrome Before and After Recovery (cf Figure 21)

ride diet, and/or ammonium chloride, the steroid therapy may have to be modified.

Because of the possible danger of continued estrogenic medication leading to cancer, it has been our practice to interrupt the medication for 7 to 14 days every 4 to 6 weeks, even though the uterus is out. An examination of the vaginal smear every 6 months provides a further safeguard. If the uterus is in, a record should be kept of the vaginal bleeding; any bleeding not according to plan (that is, not following estrogen or progesterone withdrawal) should promptly be investigated further.

Since osteoporosis is a deficiency in bone matrix protoplasm, a high protein diet is probably indicated, since it is not a disease of calcium and phosphorus metabolism, excessively high intakes of these minerals and of vitamin D are probably not indicated. Pro-

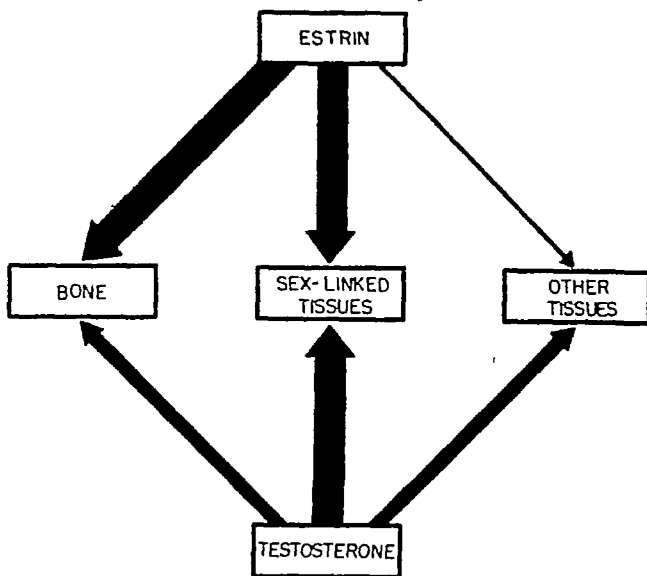


Fig 23 Diagram to Illustrate One Possible Theory of the Mode of Action of Steroid Hormone in Osteoporosis

For discussion, see text

longed immobilization should, of course, be avoided if possible, because of the danger of superimposed atrophy of disuse

Conclusions from Clinical Studies

1. Response
   A large number of cases (treated with estrogen alone and in combination with androgen) as a group have responded well.
   The pain in spine and other bones is considerably or completely relieved in weeks to months.
   The body weight is frequently increased.
   The skin appears to be thicker.
   The strength is increased.
   The general well-being is much improved

2. Dosage
   Estrogen.
     Oral
       Stilbestrol 0.5 to 1 mg daily.
       Premarin 1.25 mg two or three times daily.
     Injection
       Estradiol Benzoate 1 66 to 3.32 mg. three times weekly.
       Estradiol Dipropionate 5 mg. weekly.
       Intermittent therapy always to avoid bleeding not according to plan and overstimulation of breast, uterus, etc Give estrogen for 4-6 weeks, then skip 7 to 10 days and repeat
   Androgen:
     Oral
       Methyl testosterone 10 to 20 mg. daily.
     Injection
       Testosterone propionate 10 to 20 mg. weekly.
     Pellets
       Testosterone 75 mg. every three months.
       Give androgen continuously for first 3 months at least.

3. Other Therapy
   1 If edema develops
      a. cut down sodium intake
      b. give ammonium chloride
      c. reduce steroid dosage
   2 High protein diet
   3. Force fluids.
   4. Avoid excess calcium or Vitamin D.
   5 Avoid excessive immobilization.
   6 Support for spine corset or brace.
   7. Teach patient correct use of muscles to spare back.

A Theory of the Action of Steroid Hormones on Osteoporosis

The mode of action of the steroid hormones on osteoporosis is not known. One of the many possible theories that could be advanced is illustrated in Fig 23 This theory can be stated as follows Both estrogen and androgen are tissue stimulants Both have a marked effect on sex-linked tissues Both also have an effect on all other body tissues, but that of androgen is somewhat greater than that of estrogen As far as nitrogen is concerned, bone is just an ordinary body tissue. As far as estrogen is concerned, bone is a sex-linked tissue because it provides a storehouse for calcium to be used by the mother in supplying the fetus during pregnancy Hence, estrogen has a greater effect on bone than has androgen

REFERENCES

1 Reifenstein, E C, Jr, and Albright, F The Metabolic Effects of Steroid Hormones in Osteoporosis, J. Clin Invest 26 24-56 (Jan 1947)

2 Reifenstein, E C, Jr., Albright, F, and Wells, S. L The Accumulation, Interpretation and Presentation of Data Pertaining to Metabolic Balance, Notably those of Calcium, Phosphorus, and Nitrogen, J. Clin Endocrinol, 5 357-395 (Nov 1945)

3 Reifenstein, E C, Jr, and Albright, F Paget's Disease its Pathologic Physiology and the Importance of This in the Complications Arising from Fracture and Immobilization New Eng J Med, 231 343-355 (Sept 7, 1944).

4 Reifenstein, E C, Jr, and Albright F Conferences on the Metabolic Aspects of Convalescence, Particularly Transactions of the Twelfth Meeting, February 4-5, 1946, p 152-167 Distributed by the Josiah Macy, Jr, Foundation, New York

5 Albright, F The Effect of Hormones on Osteogenesis in Man, Recent Progress in Hormone Research, Vol 1, p 293-353, New York, Academic Press, 1947.

6. Albright, F, and Reifenstein, E C, Jr. The Parathyroid Glands and Metabolic Bone Disease Selected Studies (Monograph in Preparation)

7 Albright, F, Bloomberg, E, and Smith, P H · Post-Menopausal Osteoporosis Tr. Assoc Am Phys, 55 298 (1940)

8 Albright, F, Smith, P H, and Richardson, A. M Post-Menopausal Osteoporosis Its Clinical Features J A M A, 116, 2465 (1941)

9 Albright, F, Burnett, C H, Cope, O., and Parson, W Acute Atrophy of Bone (Osteoporosis) Simulating Hyperparathyroidism J Clin. Endocrinol, 1 711 (1941)

10 Albright, F, Parson, W, and Bloomberg, E.. Cushing's Syndrome Interpreted as Hyperadrenocorticism Leading to Hypergluconeogenesis Results of Treatment with Testosterone Propionate J. Clin Endocrinol, 1 375 (1941)

11 Albright, F Cushing's Syndrome Its Pathological Physiology, Its Relationship to the Adrenogenital Syndrome, and Its Connection with the Problem of the Reaction of the Body to Injurious Agents ("Alarm Reaction" of Selye) The Harvey Lecture Series, 38 123 (1942-1943).

INTERPRETATION OF LABORATORY DATA IN SKELETAL DISORDERS

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and
Edmund B Flink, Ph.D., M.D.

The skeleton is to be recognized as a participant in many metabolic processes It is, on this account, subject to change when certain metabolic processes are altered quantitatively or qualitatively. Several instances of the dependence of bone for its normal constitution and character on nutritional and hormonal influences have been reviewed by Dr. McLean and Dr. Reifenstein

We also recognize several disorders of bone which alter the metabolic milieu and produce alterations from the normal in the composition of the blood and urine. These latter sorts of biochemical changes are frequently of diagnostic value in bone disease For this reason an attempt will be made to review the clinical usefulness of calcium, phosphorus, and phosphatase studies While it is true that by the use of the history, x-ray, and physical examination a careful clinician can establish the diagnosis of a majority of bone diseases, in certain cases it is possible to arrive at a correct diagnosis only by a correlation of all procurable data, including laboratory studies. It is the purpose of this paper so to present this topic as to afford an understanding of the physiological basis for departures from the normal limits of laboratory data and thus to avoid the necessity for the empirical application of laboratory studies.

Table I shows the consensus as to the normal variations of total calcium and acid-soluble phosphorus in the serum. It will be noted that there is a tendency for each to decrease with increasing age. The red blood corpuscles possess a high acid-soluble phosphorus content which contaminates the serum if the red cells are hemolyzed. It is therefore necessary to

Table I

Normal Values Serum Calcium
and Serum Phosphorus

	Infants	Children	Adults
Calcium (mg per 100 cc)	10 5 - 12 0	10 0 - 11.5	9.5 - 10 5
Phosphorus (mg. per 100 cc)	5 5 - 6.5	4.5 - 5.5	2.5 - 4.0

employ serum completely free of hemolyzed cells for the determination of serum phosphorus.

The determination of total serum calcium alone is an incomplete appraisal of the physiological normality of blood calcium. The elegant work of Drs. F. C. McLean and A. B. Hastings[1,2] has demonstrated the existence of two forms of serum calcium, one free and ionized and the other bound to protein and unionized. These workers were able to make direct determinations of the ionized calcium in serum by employing the frog's heart as an indicator and found the ionized calcium normally to be 4.25 to 5.25 mg. per 100 cc. By the use of the deductions made from this study, we are, fortunately, able to calculate the calcium ion concentration from the results of two relatively simple chemical analyses, total serum calcium and total serum protein In order to make this calculation one uses the nomogram devised by McLean and Hastings[3] and reproduced as Fig. 1. For example, if the total serum protein is 8.0 grams per 100 cc. and the total calcium is 11 0 mg. per 100 cc. the lines indicating these results meet at the diagonal line which indicates that the ionized calcium is 4.5 mg. per 100 cc.

The serum-ionized calcium is characteristically high in hyperparathyroidism and in vitamin D overdosage, it is low in hypoparathyroidism and in hyperphosphatemia of uremia. In nearly all other conditions the calcium ion concentration of the serum is normal.[3]

A most important point of general application of the work of McLean and Hastings is to emphasize the dependence of total serum calcium on the level of serum protein. In order to make this point clear the protein-total calcium relationship has been diagrammatically indicated in Fig. 2 by plotting a curve from

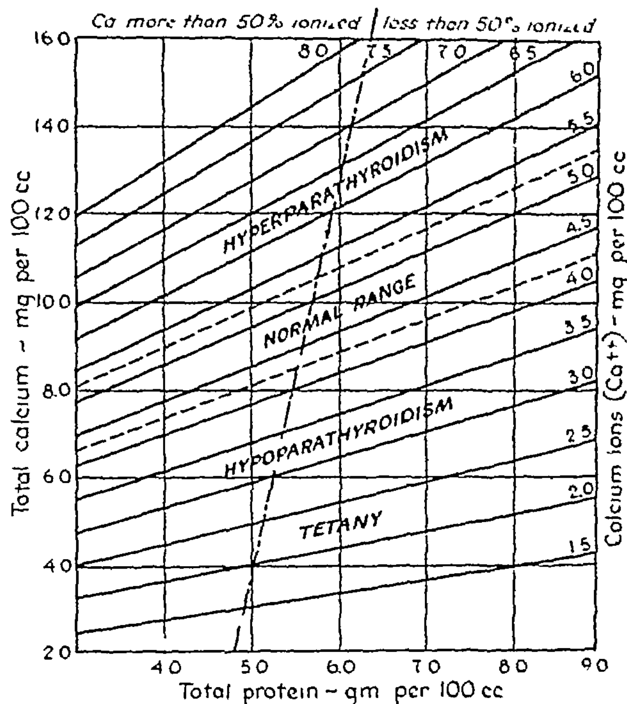


Fig 1 Chart for calculation of calcium ion concentration from total protein and total calcium of serum or plasma.

points read from the nomogram of McLean and Hastings. With a fixed value of ionic calcium in the normal range one can see that a hyperproteinemia must be accompanied by an elevated total serum calcium and, contrarywise, low levels of total serum protein are associated with low levels of total serum calcium. Failure to recognize the protein-total calcium relationship would sometimes obscure an actual hypercalcemia in cases of hyperparathyroidism with low levels of plasma protein. In other instances a lack of appreciation of the protein-calcium relationship might lead to the false assumption of a hypocalcemia.

Table II indicates some basic facts with regard to the phosphatase enzymes. These enzymes cause the hydrolysis of esters of phosphoric acid in the body as their physiological function, and the same effect is made the basis of phosphatase-activity determinations in the laboratory. The two most generally used methods, the Bodansky and King-Armstrong procedures, differ primarily in the phosphoric ester substrate employed in the activity measurements In the Bodansky[4,5] procedure glycerolphosphate is hydrolyzed, and the phos-

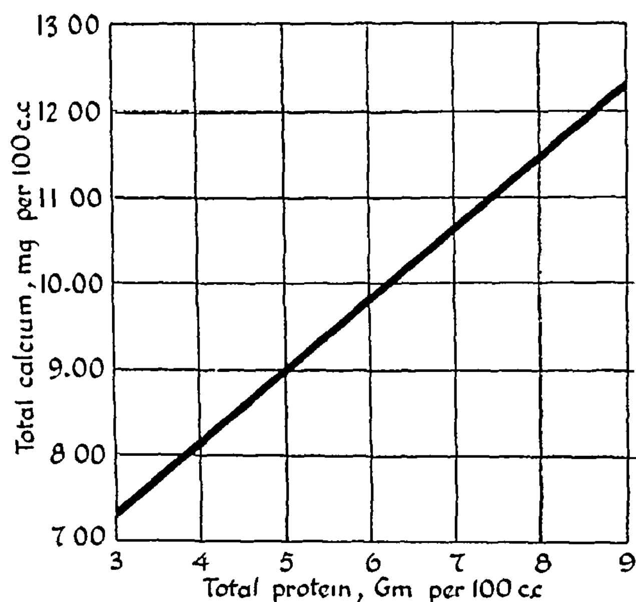


Fig 2 Relation of total calcium to total protein content of serum or plasma

phate which is liberated is determined The King-Armstrong[6] method utilizes phenyl phosphate as a substrate, and the liberated phenol is determined.

On the basis of activity of phosphatases at different pH ranges three types of phosphatase can be recognized. An alkaline phosphatase with an optimum pH at 9.3 is found in calcifying cartilage and bone The same enzyme is present in blood serum because it diffuses from the sites of its formation. This enzyme in bone is elaborated by the osteoblasts, and an increased activity of this enzyme in the serum is an indication of bone formation or at least an effort at bone formation. This statement has an exception only in the case of regurgitation jaundiee, in which no bone disturbances are present, but which is associated with an increased alkaline phosphatase activity in the serum.

The other two phosphatases of importance in the present discussion are called acid phosphatases because the pH of their optimal activity is on the acid side of the neutrality point. The erythrocyte phosphatase is important only because it makes it necessary to use hemolysis-free serum in the determination of phosphatase in serum. The prostatic phosphatase is important in relation to cancer of that organ, which relationship will be referred to presently.

Table III shows the accepted normal limits of variation of alkaline and acid phosphatase in the serum. The results in units obtained with the King-Armstrong procedure are higher than those of the Bodansky method chiefly because of the difference in substrate. On this account the King-Armstrong procedure is usually more convenient, especially in studies of acid phosphatase activity.

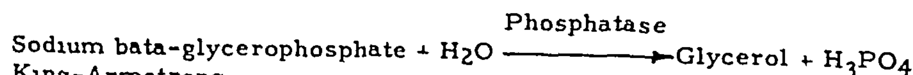
In Table IV a presentation is made of the diseases in which the alkaline phosphatase of the serum is elevated regularly or frequently enough to be of importance in differential diagnosis.[7] It will be noted, that, with the exception of regurgitation jaundice and late pregnancy, all are diseases which primarily

Table II

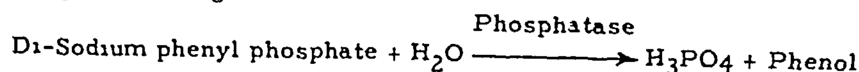
Characterization of Phosphatases

I. Reactions employed in determination (hydrolysis of an ester of phosphoric acid)

A Bodansky



B. King-Armstrong



II Types

A Alkaline

1 Optimum pH 9.3 Calcifying cartilage, bone, blood serum, intestine, kidney, spleen

B Acid

1 Optimum pH 6 Mammalian erythrocytes

2 Optimum pH 5 Prostate, prostatic metastases, serum, spleen, etc

Table III

Normal Values for Serum Phosphatase
Units per 100 cc Serum

	Acid (pH 5 0) Adults	Alkaline (pH 8 6 - 9.3) Adults	Alkaline (pH 8 6 - 9.3) Children
Bodansky*	0.0 - 0 4	1.5 - 4 0	5 0 - 12.0
King-Armstrong**		3 7 - 13 1	15 0 - 20.0
King-Armstrong** (Gutman's Modification)	0 3 - 3 25		

*Substrate - sodium B-glycerophosphate (unit is based on 1 mg P)
**Substrate - di-sodium phenylphosphate (unit is based on 1 mg Phenol)

or secondarily involve bone With regard to pregnancy, it is possible that the elevated alkaline phosphatase is the result of an attempt of the skeleton to repair its depleted state caused by the fetal demands for minerals

The premise has been made that the alkaline phosphatase activity in the nonjaundiced individual depends upon the level of osteoblastic activity This premise helps us to understand why in Paget's disease of bone, in which bone formation and bone destruction proceed simultaneously, the phosphatase activity is uniformly elevated In hyperparathyroidism it would appear at first glance that the degree of phosphatase activity is correlated with the degree of decalcification as evidenced by the x-ray However, in the usual case of this disease a vigorous, but unsuccessful, attempt at bone repair occurs Serum alkaline phosphatase is increased in rickets and osteomalacia, in which there is an attempt at bone formation and an abundance of osteoid tissue is present. Multiple myeloma is characterized primarily by bone destruction, with only minimal attempt at repair, and as a result the serum alkaline phosphatase is normal

Studies of calcium excretion are frequently of aid in the diagnosis of bone disorders, but because complete balance studies require special facilities and are laborious, they are not as commonly employed as they deserve to be As a substitute for the more complete balance studies, it is frequently useful to determine the quantity of calcium excreted in the urine when the patient is given a calcium-restricted neutral ash diet.[8] The normal individual excretes on such a regimen less than 100 mg. of calcium in the urine, but in hyperparathyroidism and hyperthyroidism or in any condition associated with a rapid decalcification of the skeleton upwards of 200 mg. of calcium per day are excreted by way of the urine

Table V is a summary of the differential diagnostic features of diseases with disturbed calcium and phosphorus metabolism

Hyperparathyroidism is frequently a hidden

Table IV

Diseases in Which Alkaline Phosphatase May be Elevated

Rickets	Osteomalacia
Paget's disease of bone	Renal Rickets (or renal osteodystrophy)
Hyperparathyroidism with osteitis fibrosa cystica generalisata	Neurofibromatosis of bone or osteitis fibrosa cystica diffusa
Osteogenic sarcoma	Carcinomatous metastases to bones
Hodgkin's (and other lymphoblastoma) involving bone	Boeck's sarcoid
Jaundice of regurgitation type	Carcinoma of the prostate with metastases
Last trimester of pregnancy	Extensive fractures in healing phase

Diseases With Abnormally Low Phosphatase

Cretinism	Scurvy

Table V

Differential Diagnostic Features of Diseases With Disturbed Calcium and Phosphorus Metabolism

Condition	Serum			Urine		Feces	Remarks
	Ca	P	p^{tase}	Ca	P	Ca	
Hyperparathyroidism	NHH	LL.	NHH	HHH	.HHH	N	
Hyperthyroidism	N	N	NH	.HHH	.HHH	.HH	
Paget's Disease	N	N	.HHH	NH	N	.	
Multiple Myeloma	NH	N	N	NH	NH	.	
Rickets	LN	LL.	HHH	L.	L.	.	
Osteomalacia	LN	L.	.HH	L.	L.	L	
Idiopath Steatorrhea	LLN	LN	NH	L.	L.	HH	
Renal Rickets	LN	.HH	.H	L.	L.	.	
Osteogenic Sarcoma	N	N	NH	N	N		
Metastatic Carcinoma	NH	N	NH	.H	.H		
Prostatic Carcinoma	NH	N	HH	.	.	.	Acid p^{tase} up
Neurofibromatosis	NH	LN	NH	.	.		
Uremia	LL.	.HH	N	.	LL		
Regurgit'n Jaundice	N	N	.HHH	.	.	.	
Hyperproteinemia	.H	N	.	.	.	.	Ca function
Hypoproteinemia	L.	N	.	.		.	normal
Acidosis	N	N		.	.	.	Ca ions up
Alkalosis	N	N	.	.		.	Ca ions down
Hypoparathyroidism	LLL	HH	N	LLL.	L		
High Vit. D. Therapy	.HH	HH	N	.HH	.HH	.	

Schema N - normal. LL - decreased HH - increased. The number of H's or L's indicates roughly the extent of increase or decrease

disease and its consistent recognition depends upon x-ray examination of all patients with musculo-skeletal symptoms and of those who have symptoms of renal calculi or present a history of mild polyuria and polydipsia The bone lesions in this condition have received sufficient emphasis to be familiar to all What is less often recalled is the serious renal damage which occurs in untreated hyperparathyroidism of even a mild degree

The characteristic biochemical findings in hyperparathyroidism are an increased calcium-ion content of the serum, which is reflected in an elevated total calcium of the serum and a decreased serum inorganic phosphorus. If the patient has a decreased content of protein in his plasma the total calcium may be within the usually accepted range, but, as has been indicated previously, a normal calcium associated with a low plasma protein is in fact a hypercalcemia The phosphatase is elevated and the excretion of calcium and phosphorus in the urine is usually increased. The renal lesions which may be of several varieties, due to stone formation or to calcification of the kidney parenchyma, are caused by the high concentration of calcium and phosphate in the urine.

The phosphatase activity has a special significance in hyperparathyroidism since the probability of occurrence of postoperative tetany following removal of a parathyroid adenoma is much greater in patients with high phosphatase values than in those with nearly normal phosphatase activity. The reason is, of course, that phosphatase activity is a measure of the avidity with which bone will retain calcium when the parathyroid activity is made normal by surgical intervention.

Chronic hyperthyroidism is an important cause of osteoporosis because of the increased excretion of calcium in the urine and feces, and in such patients it is difficult to maintain a positive calcium balance during the active phases of the disease.

Paget's disease is never generalized. It is chronic and tends to be progressive. It is not infrequently accidentally discovered as a result of roentgenograms taken for other reasons, although bone pain, pathological frac-

tures or deformities of the skull may call attention to the disorder. Reifenstein and Albright have demonstrated the necessity for as little immobilization as possible when fractures involved in the disease process must be treated If the immobilization is unduly prolonged an acute atrophy of bone occurs, and consequently, upon the increased excretion of mineral ions in the urine, serious renal complications may arise In Paget's disease the serum calcium and phosphorus are normal but the phosphatase is usually markedly elevated As previously stated, this latter finding is due to the fact that in Paget's disease bone formation, while of a bizzare character, occurs simultaneously with bone resorption

Multiple myeloma, as has already been indicated, is a disease with normal or only slightly elevated phosphatase activity The serum calcium may be elevated secondarily to the hyperproteinemia. Aside from the calcium, phosphorus, and phosphatase findings, there are several other important laboratory aids in the diagnosis of multiple myeloma An elevated erythrocyte sedimentation rate in the absence of infection and in a patient who complains of skeletal symptoms or gives evidence of malignancy should make one suspect multiple myeloma Bence-Jones protein appears in the urine of many patients but is frequently absent and is not pathognomonic of multiple myeloma Hyperglobulinemia occurs frequently and may be extreme The diagnosis is usually established by means of sternal marrow biopsy

The diagnosis of active rickets is to be entertained, of course, only in the case of infants Here the x-ray findings correlated with the low serum phosphorus and high phosphatase activity are of diagnostic importance. Many clinicians have such a firm fixation as to the diagnostic value of the serum calcium level that they request a total calcium determination as the sole laboratory study in cases of suspected rickets, when, in fact, the blood calcium is usually normal in these cases The blood calcium falls only when the cases are partially or incorrectly or intermittently treated, causing a rapid calcification of the large volume of osteoid tissue

Idiopathic steatorrhea may result in an interference with calcium absorption so as to produce hypocalcemia and tetany. Osteomalacia, which is classically due to severe calcium and vitamin D deprivation, usually occurs in this country as a result of chronic mild steatorrhea Such cases of osteomalacia usually have the onset of their steatorrhea before adult life is reached. The laboratory findings of osteomalacia are similar to those of rickets except that the degree of the disturbances may not be as severe as those of rickets and in osteomalacia secondary to diminished calcium absorption of steatorrhea, the serum calcium tends to be low

Renal osteodystrophy or renal rickets occurs in children with chronic renal insufficiency from any cause which is severe enough to result in the retention of nonprotein nitrogenous substances and inorganic acid radicals such as phosphate and sulphate The most important feature of the chemical findings is the retention of phosphate with a normal or low serum calcium and a moderately elevated phosphatase activity In chronic uremia, both in children and in adults, a form of parathyroid hyperplasia may occur, producing a secondary hyperparathyroidism with osteitis fibrosa cystica Presumably the high phosphate level of the serum stimulates hyperplasia of the parathyroids and this in turn causes secondary hyperparathyroidism The high serum phosphate serves to distinguish secondary hyperparathyroidism due to renal disease from primary hyperparathyroidism. However, when renal insufficiency results from primary hyperparathyroidism the characteristic high calcium, low phosphorus of primary hyperparathyroidism may be converted to a normal calcium and high phosphorus. It is thus difficult to distinguish primary hyperparathyroidism with severe renal failure from the secondary hyperparathyroidism of primary renal insufficiency This differential diagnosis must be established from the history and the degree of skeletal changes

Nothing in particular needs to be said with regard to the essentially normal biochemical findings in osteogenic sarcoma It is evident that the laboratory findings in carcinomatosis overlap those of multiple myeloma, and widespread metastases to bone are apt to be confused with multiple myeloma or even hyperparathyroidism These differentiations have to be made on other grounds Neurofibromatosis (osteitis fibrosa cystica disseminata) may present x-ray findings which superficially resemble those of hyperparathyroidism, and the lab-

oratory data in these conditions may be quite similar. This differential diagnosis should be made by the demonstration of the stigmata of neurofibromatosis.

Prostatic tissue has a very high acid phosphatase content and unless a prostatic carcinoma is very undifferentiated, it and its metastases, also produce and release to the blood increased amounts of this enzyme. The metastases to bone stimulate an increased osteoblastic activity in their environment, which accounts for the frequently elevated alkaline phosphatase seen in such cases.

The disturbances below the horizontal line in Table V are not diseases which significantly affect the skeleton Because alterations from the normal are seen in serum calcium, phosphorus, and phosphatase, a few words with regard to these disorders may be of interest

The elevated inorganic phosphorus of the serum in uremia may alter the physio-chemical balance of calcium and phosphorus so as to cause a reciprocal decrease in total calcium of the serum. Regurgitation jaundice, as has already been mentioned, frequently results in an elevated alkaline phosphatase The interrelationship of protein and total calcium has already been discussed in relation to hyperproteinemia and hypoproteinemia. In acidosis and alkalosis the total calcium is normal but the calcium ion concentration is elevated in acidosis and decreased in alkalosis

Hypoparathyrodism is not an orthopedic disease, but an appreciation of the physiological disturbances occurring in this condition aids the understanding of other disturbances of mineral metabolism. Cases of manifest tetany occurring in an individual who has had a thyroidectomy will, of course, be easily recognized In these individuals the total calcium and calcium ion concentration of the serum will be found to be depressed and the inorganic phosphorus of the serum will be elevated. Delirium occurs frequently in acute hypoparathyroidism, and any patient who becomes delirious after thyroidectomy should be carefully examined for signs of hypocalcemia. In these patients it is important to exclude chronic renal insufficiency in the differential diagnosis.

The rarer cases of spontaneous hypoparathyroidism, especially those in which tetany is only latent, are more difficult to recognize, both clinically and by laboratory examinations. Such patients have been treated as neurotics or malingerers The Trousseau and Chvostek signs are useful in recognizing latent tetany. Careful studies of total calcium and calcium ion concentration in the serum will usually point to the correct diagnosis When tetany occurs it must be treated promptly, since laryngeal or diaphragmatic spasm may be fatal The treatment of hypocalcemic tetany is a medical problem and has no place in this discussion.

REFERENCES

1 McLean, F.C., and Hastings, A.B. J Biol. Chem 107 337 (1934)
2. McLean, F.C , and Hastings, A.B. J. Biol Chem. 108 285 (1935).
3 McLean, F.C., and Hastings, A. B. Am J Med. Sci. 189 601 (1935)
4 Bodansky, A. J. Biol. Chem. 99 197 (1932).
5. Bodansky, A. J Biol Chem. 101 93 (1933).
6. King, E. J., and Armstrong, A.R. Can. Med. Assoc J. 31 376 (1934).
7. Sunderman, F. W. Am. J. Clinic. Path. 12 404 (1942).
8. Albright, F., Sulkowitch, H. W., and Bloomberg, E Am. J. Med. Sci. 193 800 (1937).

Course No. 5

NECK LESIONS

Lecturers

Ralph Ghormley, M D , "Shoulder and Arm Pain"

Kendall B Corbin, M D , "The Anatomic Basis for the More Common Types of Mechanical Brachial Neuritis"

Arthur G Davis, M D , "The Relation of Upper Extremity Pain to Cervical Injuries"

Carl E Badgley, M.D , "Brachialgia"

SHOULDER AND ARM PAIN

Ralph Ghormley, M.D.

KNOWLEDGE OF THE syndrome or syndromes of arm pain and their relation to lesions of the cervical part of the spinal column, shoulder, and brachial plexus has improved slowly compared to knowledge of the sciatic syndrome.

Painful lesions of the shoulder commonly involve the capsule or musculotendinous cuff Less frequently encountered painful lesions are those of the head of the humerus and of the scapular structures The most common painful lesions of the shoulder are 1) frozen shoulder, capsulitis, tendinitis, peritendinitis, or periarthritis, 2) calcified subdeltoid bursitis, subdeltoid bursitis or degeneration of supraspinatus tendon, and 3) tears of the supraspinatus tendon and of the musculotendinous cuff.

Codman[1] must be credited with describing incomplete and complete tears of the supraspinatus tendon He stated that among the lesions of the shoulder (exclusive of tumors, fractures, and infections), calcified deposits, tendinitis, partial and complete rupture of the supraspinatus tendon have about an equal incidence.

Review of the writings of various authors reveals a varied opinion as to the incidence of lesions of the musculotendinous cuff Although definite figures are not available on the incidence of each of these lesions, Codman's estimate may be used for the basis

Certain facts have come to be accepted concerning painful lesions of the shoulder 1) The supraspinatus tendon in a fair number of persons tends to degenerate and the frequency of this occurrence increases with age. 2) There is general agreement that the so-called periarthritis or frozen shoulder has its inception in some type of inflammation of the tendon or tendon sheath. 3) Calcification in bursae probably results from accumulation of degenerated tendinous material. 4) Ruptures of the supraspinatus tendon are fairly common in older persons They may cause severe symptoms in some cases and require surgical treatment. 5) Among younger persons following trauma, tears of the musculotendinous cuff are much more severe and serious than among older persons Surgical repair is indicated whenever possible but in the more severe tears arthrodesis may be necessary to relieve the painful symptoms

Lesions that have not been mentioned which may cause painful shoulder are· 1) tumors and infections of the scapula or head of the humerus, 2) lesions of the clavicle, 3) lesions of the acromioclavicular joint, 4) tumors of the soft tissues about the shoulder, 5) tuberculous and other types of arthritic involvement of the shoulder, and 6) fractures or fracture dislocations of the shoulder

The differential diagnosis of lesions of the shoulder from lesions of the cervical portion of the spinal column or brachial plexus often is a difficult problem It must be remembered in cases of pain in the shoulder or arm that lesions of both the cervical part of the spinal column and shoulder joint may be present The most important point in differential diagnosis is the presence of local signs in the shoulder, that is both active and passive limitation of motion, either diffuse or localized tenderness, atrophy of variable severity, and swelling or local heat in an occasional case of acute bursitis In cases in which the shoulder joint is free of all of these signs, the primary cause of the trouble usually is elsewhere.

Whenever shoulder and arm pain is considered, it must be remembered that a number of lesions of the chest may cause this pain also Of these I shall mention cardiac pain, which may be referred to the arm first. According to Smith[2] the most important feature of such pain is that it must be produced by effort. It occurs during effort and not afterward.

Lesions of the superior pulmonary sulcus adjacent to the brachial plexus or sympathetic chain also may cause pain of the arm.[3] Pancoast[4] described a syndrome including roentgenographic shadows of the tumor at the apex of the lung, a neuritic type of pain of the arm, atrophy of the muscles of the hand and arm, and Horner's syndrome. This syndrome is not a distinct clinical entity since neurofibromas or inflammatory lesions as well as malignant lesions may cause it. Pain from lesions of the esophagus is rarely referred to the arm. Diaphragmatic lesions, however, may cause pain in the arm This is particularly true of lesions of the central portion of the diaphragm, such as esophageal hiatal hernias. Diaphragmatic pleurisy also may cause pain in the shoulder and arm

REFERENCES

1 Codman, E. A The Shoulder, Rupture of the Supraspinatus Tendon and Other Lesions in or about the Subacromial Bursa Boston, Thomas Todd Company, 1934, 513 pp.

2. Smith, H L. Arm Pain Due to Heart Disease. S. Clin North America. 26 804-805 (Aug., 1946)

3 Olsen, A M The Role of Thoracic Disease in the Production of Arm Pain. S. Clin. North America. 26 801-803 (Aug., 1946).

4. Pancoast, H. K. Superior Pulmonary Sulcus Tumor, Tumor Characterized by Pain, Horner's Syndrome, Destruction of Bone and Atrophy of Hand Muscles. J. A M. A. 99·1391-1396 (Oct. 22, 1932).

THE ANATOMIC BASIS FOR THE MORE COMMON TYPES OF MECHANICAL BRACHIAL NEURITIS

Kendall B Corbin, M.D

Today, most clinicians who concern themselves with that disorder known as brachial neuritis would agree, I feel certain, that in the vast majority of cases the neurologic signs or symptoms are the result of mechanical, intraspinal, or extraspinal compression of the components of the brachial plexus. These disorders, therefore, would be classified as mechanical neuritides, rather than placed in that much smaller group of toxic-infectious lesions of nerves This concept is not new but frequently the physician who is not aware of the numerous possibilities for compression which exist in the region of the neck, shoulders, and arms and who does not make certain fundamental tests, including adequate roentgenographic studies, may resort to the nonspecific diagnosis of brachial neuritis. A knowledge of the functional anatomy of this region is, therefore, indispensable to a proper understanding of the mechanisms by which the cervical roots and plexus may suffer trauma by functional or anatomic variations from the usual.

The seven cervical vertebrae enclose in their formed canal the eight cervical segments of the spinal cord and a variable portion of the

first thoracic segment From the level of the third cervical segment downward the cord increases in size and attains its greatest diameter and circumference at approximately the sixth cervical segment, which lies opposite the lower part of the fifth cervical vertebra. The cervical enlargement extends approximately to the level of the second thoracic segment of the cord.

This portion of the spinal cord, like the remainder of the central nervous system, is enclosed by the meningeal membranes (Fig. 1). The pia-arachnoid encloses the subarachnoid space, the latter contains the cerebrospinal fluid. Immediately exterior to the arachnoid lies the tough, fibrous dura mater, suspended from above at the foramen magnum, where it is continuous with intracranial dura and periosteum. Thus the spinal epidural space terminates superiorly at the foramen magnum. The spinal dura is more or less completely surrounded by epidural fat and venous plexuses and lies completely free in the vertebral canal except for fibrous strands along its anterior aspect which attach it to the posterior longitudinal ligament, rather securely in the upper cervical region, loosely below. The spinal cord, in addition to its attachment above to the brain stem, is supported by laterally placed denticulate ligaments, extending between the pia and the dura mater

The spinal cord thus is relatively well protected in a bed of cerebrospinal fluid, bounded be arachnoid and dura. External to the latter lies another relatively fluid mass, the epidural veins and fat, between dura and the investing membranes of the spinal canal.

The third to eighth cervical roots pass laterally and anteriorly to enter their respective intervertebral foramina where the posterior root ganglia of each are located. At the proximal side of each ganglion the investing dura becomes firmly bound to ganglion and roots and then continues distally as the epineurium of the spinal nerves. The ganglia of the first and second cervical nerves lie on the arches of the axis and atlas (Fig. 1).

The greatest protection to the spinal cord is, of course, the canal formed by the separate vertebrae, their intervertebral disks, and their combined ligaments. On the other hand, malposition, herniation, and a shift in position of these elements, secondary to trauma or disease, cause some of the most serious of the

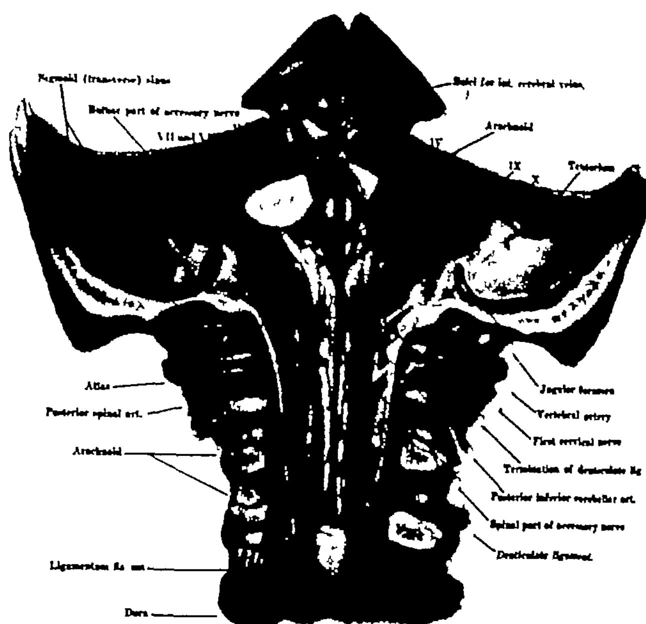


Fig 1. Dorsal view of brain stem and cervical portion of the cord, showing meningeal relationships. (From Mettler, F A. Neuro-anatomy, 1942, p. 46. By permission of the author and publisher, C V. Mosby Company)

injuries to cord and root seen in civilian life.

The seven cervical vertebrae form as a whole an exceedingly mobile segment of the spinal column. The junction of second and third cervical vertebrae, where the intervertebral disk is thin, is considered the weakest part of the spinal column, although this is not the part most liable to injury. The anterior convexity of the cervical curvature which culminates at the fifth cervical vertebra may be compared with the lumbar curvature, both represent regions in which anterior flexion causes great stress on the intervertebral disks. This fact has been mentioned by most observers in explaining the high incidence of posterior herniation of intervertebral disks at these levels. The spinal curvatures serve an important protective function in damping jolts and absorbing vibrations

A word of explanation is in order regarding the anatomic peculiarities of the cervical portion of the spinal column which frequently lead to confusion in designating the level of neural, vertebral, or intervertebral involvement. Since no disks are present between occiput and atlas or between atlas and axis, naming the disk involved by number may be misleading. The first

cervical nerve leaves the spinal canal between the atlas and the occiput, the eighth between the seventh cervical and the first thoracic vertebrae (Fig. 2). No clearly defined intervertebral foramen exists between atlas and axis so that we are left with only six specific intervertebral foramina. Thus it seems desirable in specifying the level of involvement in the cervical region, especially when referring to herniation of a disk, to name the nerve involved or to specify the numbers of the adjoining vertebrae.

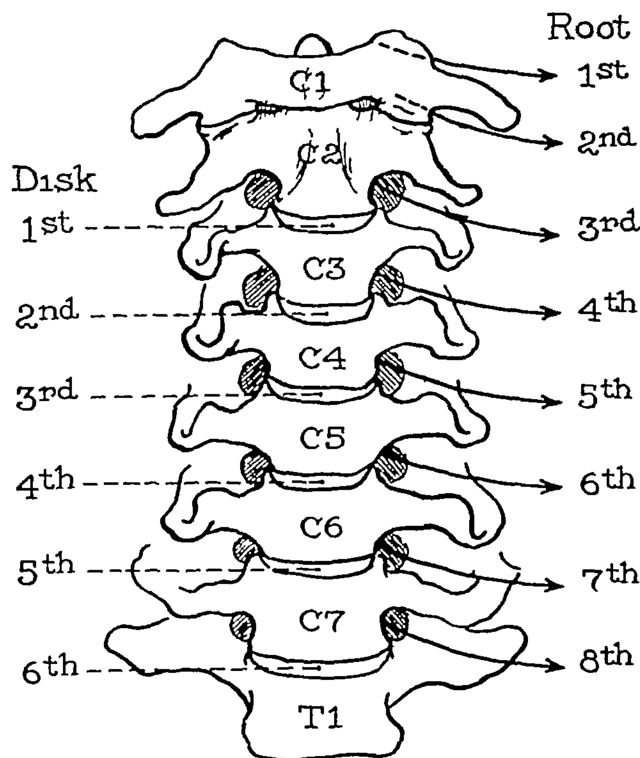


Fig. 2. Diagrammatic anterior view of cervical and first thoracic vertebrae showing exit of cervical nerves and placement of intervertebral disks (From Eaton, L M. Neurologic Causes of Pain in the Upper Extremities, with Particular Reference to Syndromes of Protruded Intervertebral Disk in the Cervical Region and Mechanical Compression of the Brachial Plexus S Clin. North America. 26 810-833 [Aug., 1946] Reprinted with permission of the author and publisher, W. B. Saunders Company.)

Several features of this portion of the spinal column deserve mention. Only the third to the sixth cervical vertebrae may be considered typical. The atlas and axis possess characteristics which distinguish them from all other vertebrae. The atlas lacks a body, possesses lateral processes with concave superior articular pits for articulation with the occipital condyles, and on the dorsal surface of the anterior arch is a circular facet for articulation with the odontoid process of the axis. The axis, is easily recognized by the projecting odontoid process which arises from the cranial surface of its body. The oval, superior articular surfaces arise from both the body and the roots of the arch. These surfaces face superiorly and laterally for articulation with the atlas, and acting with the dens as a center of rotation provide a gliding surface for rotation of the head. All of the cervical vertebrae possess foramina in their transverse processes, in which, except for the seventh vertebra, are located the vertebral artery and veins. Each foramen divides the transverse process into an anterior costal process and a posterior lateral process. The articular surfaces of the third to seventh vertebrae, since they are placed obliquely in a transverse plane, allow flexion and extension However, lateral flexion and rotation are each accompanied by some degree of the other movement [2]

The intervertebral foramina through which emerge the third to eighth cervical roots are formed by the superior and inferior notches of adjacent vertebrae Thus anteriorly lies an intervertebral disk covered by the lateral expansion of the posterior longitudinal ligament and portions of the corresponding bodies. Above and below each foramen lies the corresponding root or pedicle, and posteriorly the capsular ligament of the articular process. The latter is covered anteriorly by the ligamentum flavum. These cervical intervertebral foramina are oval in shape with their narrower diameter in the horizontal plane. The nerve roots leave the cord at more nearly right angles than do lower roots and thus lie almost directly over the corresponding intervertebral disks. The lateral protrusion of a cervical disk, therefore, may compress the root against the ligamentum flavum. lamina, pedicle, or facet without damaging the cord.

The roots and nerves occupy only approximately half of the entire space in the intervertebral foramen. For this reason some physicians have considered that radiculitis from arthritic narrowing of these spaces is highly unlikely. This is not the place to labor the point, but I am sure that compression of a root by spondylitis would best explain the clinical

picture in certain cases. It is probably true that all too often this condition is blamed for brachial radiculitis, plexitis, or neuritis when the true cause of the mechanical irritation is overlooked In this connection it should be mentioned that the bony circumference of the intervertebral foramina increase in size from above downward.

Posterior or posterolateral bulging and herniation of a cervical intervertebral disk or its nucleus pulposus is now recognized as a rather frequent cause of brachial pain. Since this syndrome will be considered at length in another paper in this symposium, I wish merely to point out several anatomic points of importance in the production of the lesion and to stress again the vulnerability of the lower part of the cervical region. Semmes and Murphey,[3] in 1943, emphasized that the anteroposterior diameters of the intervertebral foramina are smallest at the points of maximal anterior lordosis. A laterally protruding disk markedly narrows the foramen and produces root pressure, if hypertrophy of the ligamentum flavum coexists, compression of the root will occur earlier. Cervical disks are constructed more delicately and are narrower than those in the lumbar region and are required to support much less weight According to Naffziger and Boldrey,[4] the disk between the sixth and seventh cervical vertebrae is the most mobile, is the most subject to trauma, and is the most common site for compression of a nerve root of any disk in the whole spinal column.

Because of the relative shortness of the cervical nerve roots and the relatively large bulk of the cervical portion of the cord in relation to the vertebral canal, any intraspinal lesion which occupies space in this region, whether it be neoplasm, abscess, or herniated disk, will produce signs of compression of root or cord, or both, relatively early in its course

Little need be said in this paper regarding the localization of lesions in the cervical portion of the cord except to mention certain neuro-anatomic features of this portion of the cord which may be of diagnostic help. The hypothalamic-spinal pathways modifying thoracolumbar autonomic control of the cranial smooth muscles passes through the cervical cord to reach the intermediolateral cell column of the upper thoracic segments. Thus lesions low in the medulla and in the cervical part of the cord, as well as those involving the upper thoracic ventral roots, their white rami or the cervical sympathetic trunk, may cause Horner's syndrome on the side of involvement. It also should be borne in mind, when cervical lesions are considered, that the motor supply to the diaphragm, via the phrenic nerve, arises from the third, fourth and fifth cervical roots and the anterior horn cells of these segments.

How can the well-known observation that coughing, sneezing, straining and other maneuvers which increase intrathoracic, or intra-abdominal pressure aggravate root pain be explained? Eaton,[5] in 1941, demonstrated by clinical tests that the mechanism for this phenomenon lies in an engorgement of the epidural venous plexuses, secondary to the interruption of venous flow into the large abdominal and thoracic veins, and that these engorged vessels press on or stretch the irritated roots (Fig 3) The explanation commonly advanced is that the aggravation of pain

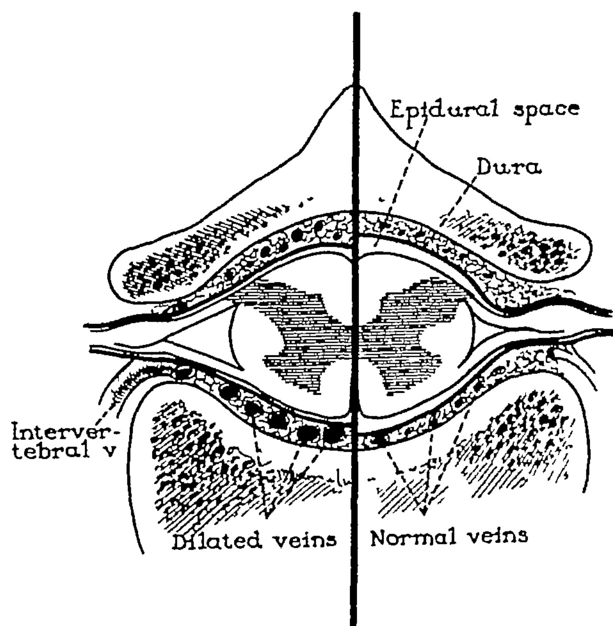


Fig. 3 Diagrammatic cross section of spinal cord, dura, and epidural space before (right) and during (left) straining maneuvers. (From Eaton, L. M. Pain Caused by *Disease Involving the Sensory Nerve* Roots [Root Pain], Its Characteristics and Mechanics of its Production J A. M A. 117 1435-1439 [Oct. 25, 1941]. Reprinted with permission of author and publisher.)

is secondary to increased pressure in the cerebrospinal fluid alone. This same author has stressed the accentuation in root pain which occurs during sleep. He attributed this to lengthening of the spinal column that takes place when the body is in the horizontal position, he attributed the lengthening to an increase in the vertical height of the intervertebral disks. Tension on an already traumatized root by such lengthening was thought by Eaton to account for the increase in pain which occurs during sleep (Fig. 4).

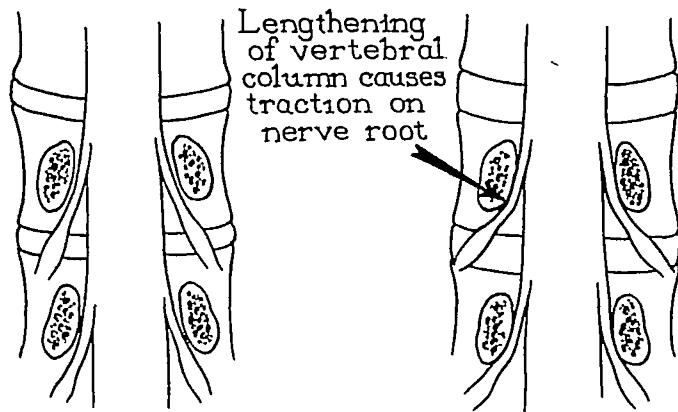


Fig. 4. Relationship of spinal roots and pedicle.

I should now like to trace the cervical roots peripherally and to mention the extraspinal sites at which they are likely to be traumatized by rather slight variations from the usual relations. Before this is done, however, a brief description of the formation of the brachial plexus will be given.

The brachial plexus arises from the anterior rami of the fifth, sixth, seventh, and eighth cervical, and the first thoracic nerves (Fig. 5). Developmentally, the cephalic roots supply the upper or pre-axial portion of the limb, the caudal roots, the postaxial border. The seventh cervical nerve may be considered the central and largest root, it supplies approximately the axis of the limb. The fifth and sixth cervical nerves supply the preaxial or radial border, and the eighth cervical and the first thoracic, the postaxial or ulnar border. When the limb is considered as a whole, it is readily seen that the fourth cervical and the second thoracic roots also contribute to its sensory supply. The contribution of either of these roots may be far more complete when the variations known as prefixed or postfixed

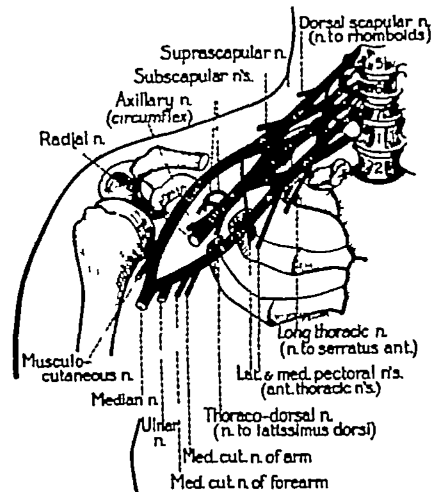


Fig. 5. Diagram of brachial plexus. (From Haymaker, Webb, and Woodhall, Barnes Peripheral Nerve Injuries, Principles of Diagnosis, 1945, p. 129. Reprinted with permission of authors and publisher, W B. Saunders Company)

plexuses occur. In the case of a postfixed plexus a large portion of the second thoracic primary ramus joins with the first thoracic to form the inferior trunk of the brachial plexus. In passing up and over the first rib this lowest root may be irritated and produce definite symptoms (Fig. 6).

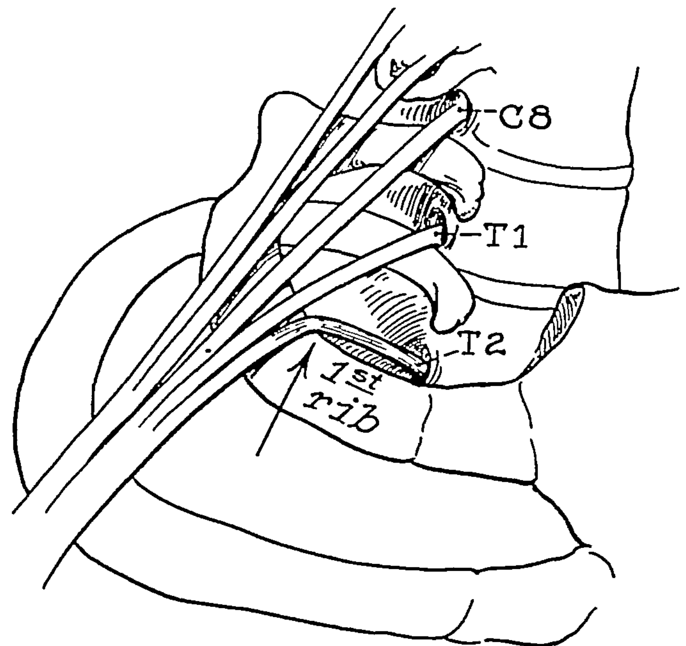


Fig. 6. Diagrammatic representation showing pressure on second thoracic root by first rib in postfixed plexus.

In the usual plexus, the upper two roots, the fifth and sixth cervical, form an upper trunk, the seventh cervical alone forms the middle trunk, and the eighth cervical and first thoracic nerves form the lower trunk. Each trunk then gives rise to three anterior and three posterior divisions. The three posterior divisions form a single posterior cord. The lateral and intermediate anterior divisions unite to form a lateral cord whereas the medial continues as such. After giving off various collateral branches, the cords terminate in the five principal nerves to the upper extremity (Fig. 5). From the lateral cord are derived the musculocutaneous and lateral root of the median nerve. The medial cord terminates as the ulnar nerve and sends off its medial root to the median nerve. The posterior cord divides into the axillary and the radial nerves

Space will not permit enumeration of the muscles supplied by each nerve or the radicular supply of each muscle. Certain basic facts should be kept in mind, however, which will be of constant aid in determining the level of root involvement in lesions of the neck and shoulder. The musculocutaneous nerve is formed by the fifth and sixth cervical nerves and in more than half of cases by the fourth cervical as well. The ulnar nerve is formed by the eighth cervical and first thoracic nerves and in half of the cases by the seventh cervical as well. The radial nerve is derived from fifth, sixth, seventh, and eighth cervical and frequently from the first thoracic nerves. The median nerve arises from the sixth, seventh, and eighth cervical and first thoracic nerves with sometimes a few fibers from the fifth cervical. Thus median and radial nerves receive fibers from practically all the roots of the plexus and the musculocutaneous and the ulnar nerves receive fibers from the cephalic and caudal segments, respectively. For further details the reader is referred to the excellent illustrations by Haymaker and Woodhall (1945).[6]

Figure 7 demonstrates the usual cutaneous sensory supply to the upper extremity, according to Haymaker and Woodhall and requires no further comment except mention of the fact that variations from this pattern are the rule rather than the exception.

The autonomic nerve supply to the upper extremity is derived entirely from the thoracolumbar system. Preganglionic cells of origin for this outflow are located in the intermediolateral cell column of the upper ten thoracic segments[7] The preganglionic fibers pass out with the ventral roots, enter the sympathetic trunk via the white rami communicantes, and ascend to synapse for the most part about the postganglionic neurons located in the stellate and the inferior and middle (when present) cervical ganglia (Fig 8). Postganglionic sym-

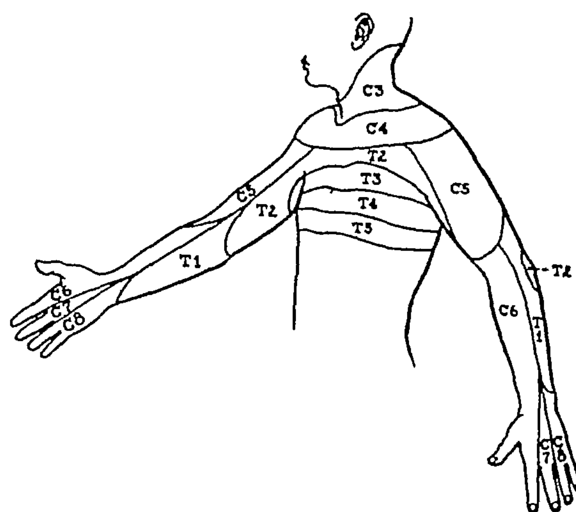


Fig 7 Dermatomic supply of cervical and upper thoracic roots (modified from Foerster)

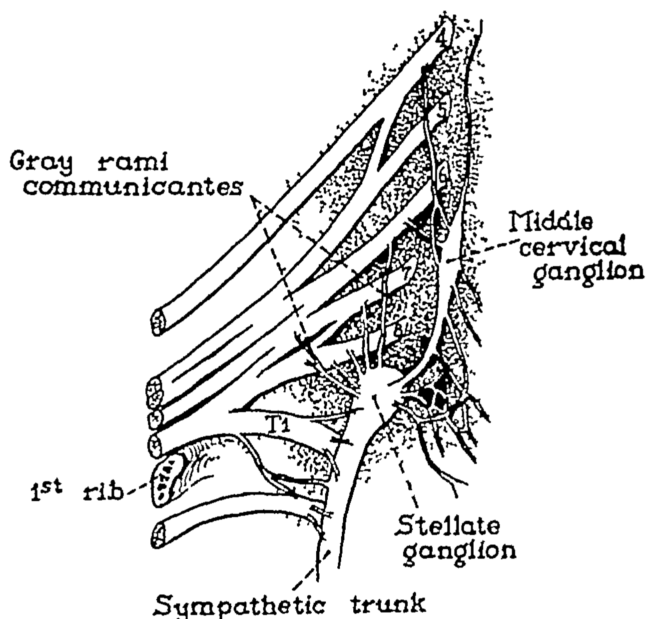


Fig. 8 Diagrammatic representation of connections between sympathetic ganglia and brachial plexus.

pathetic fibers from these ganglia join the spinal nerves from the fifth cervical to the first thoracic via gray rami and pass with these nerves to the periphery The autonomic nerve supply to the limbs does not pass peripherally along the vessels, except for relatively short distances near the termination of such nerves on the vessels.

As the cervical nerves leave the intervertebral foramina, the anterior primary rami of the third, fourth, fifth, and sixth roots pass anteriorly and laterally in the gutters grooving their respective transverse processes. In doing so, the fourth, fifth, and sixth nerves pass posteriorly to the tendons of the scalenus anterior muscle which arise in each instance from the anterior tubercles of the next higher vertebra (Fig. 9). Swank and Simeone,[8] in 1944, suggested that the roots may be compressed by these tendons against the tips of the transverse processes and named the resulting manifestations "the upper scalenus syndrome."

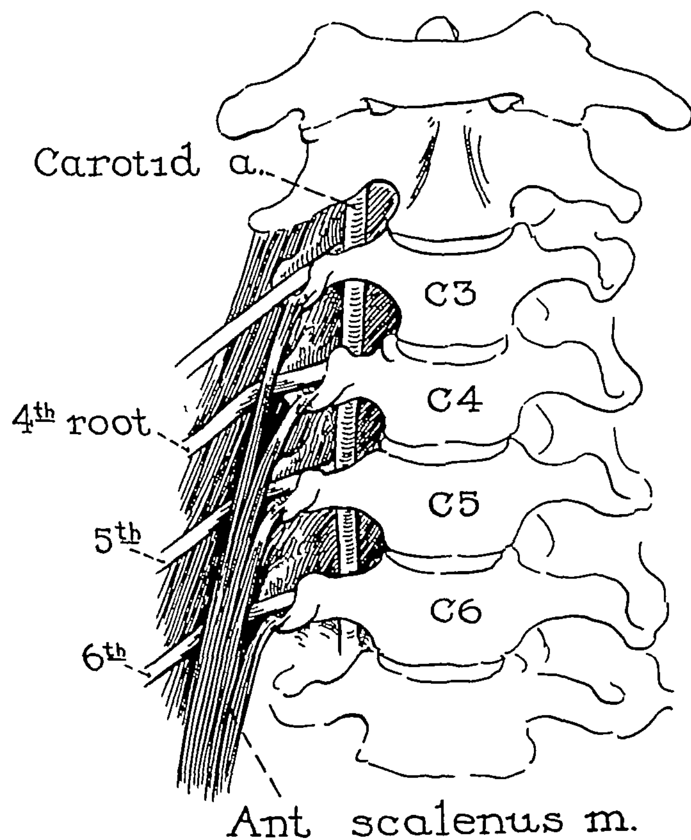


Fig. 9. Pressure on cervical roots by anterior scalenus tendons (modified from Swank and Simeone).

Between the intervertebral foramina and the axilla are several places where the roots, plexus, or accompanying vessels may be compressed by relatively minor anatomic variations. Such points of predilection, amply described by numerous authors among whom the reader is referred to Jones,[9,10] Todd,[11,12] Adson and Coffey,[13] Craig and Knepper,[14] Naffziger and Grant,[15] Eden,[16] Hill,[17] Folconer and Weddell,[18] Walshe, Jackson and Wyburn-Mason,[19] and Eaton,[1] are the following 1) the dorsal surface of a normal or rudimentary first thoracic rib; 2) the superior or anterior margin of a rib attached to the seventh cervical vertebra (less often the sixth), or the fibrous rudiment or continuation of such a rib, acting alone or in conjunction with the following, 3) the posterior margin of an abnormal, tight, hypertrophied, broad, or normal scalenus anterior muscle, 4) the region between the dorsal surface of the first rib and the inferior surface of the clavicle (or subclavius muscle) in which the subclavian artery and the lowest roots of the brachial plexus lie, and 5) the region lying behind the pectoralis minor muscle and beneath the coracoid process, during hyperabduction of the arm.

The roots of the brachial plexus emerge between the scalenus medius and scalenus anterior muscles into the posterior triangle of the neck (Fig. 10). The supraclavicular portion thus lies on the fascia of the scalenus medius and the serratus anterior muscles. The lowest root, namely, the first thoracic (occasionally the second), passes up and over the first rib to join the next higher root immediately lateral to the scalenus anterior muscle. The subclavian artery lies between the lowermost roots and this muscle. Passing laterally and downward the artery and lowermost trunk of the plexus lie between the first rib and the subclavius muscle. Grant,[20] in 1940, pointed out that it is this small muscle which protects the artery from the clavicle. The first rib is grooved in varying degrees by both the artery and nerve. The subclavian artery becomes the axillary artery at the lateral margin of the first rib. As it continues laterally it is surrounded by the three cords of the brachial plexus and passes directly behind the pectoralis minor muscle and inferior to the coracoid process.

Todd,[11] in 1912, was among the first to describe how the first thoracic nerve (and the second thoracic nerve in the postfixed plexus)

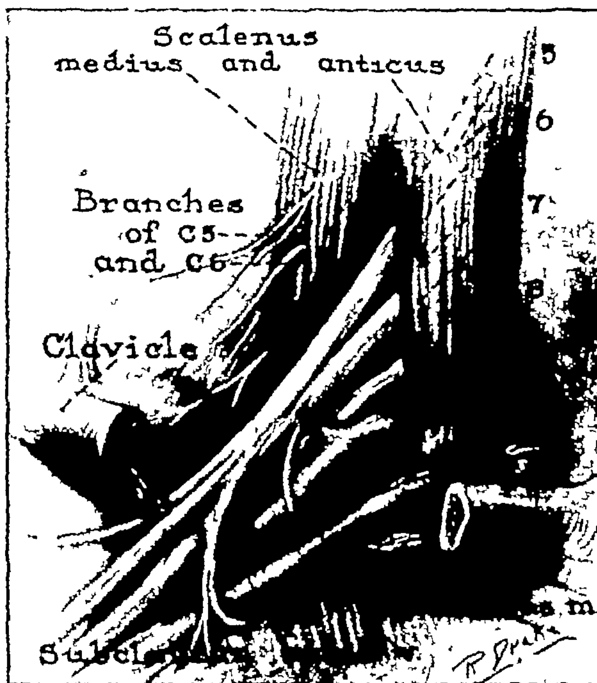


Fig 10. Diagram illustrating topography of brachial plexus in posterior triangle and upper axilla

must pass upward and cross the thoracic operculum in order to reach its destination (Fig 6). As development proceeds and the limb moves caudally from its original embryonic position, the thoracic contribution to the brachial plexus describes a U-shaped course Any influence causing excessive drooping of the shoulder then tends to stretch these nerves and produce symptoms of compression low in the plexus Both ends of the clavicle tend to descend during development In analyzing the factors which bring about descent of the inner end, secondary to descent of the first rib, Todd concluded that the most important single force was the tonic contraction of the rectus abdominis muscles acting on the sternum. The shoulder continues to descend during postnatal development, more on the dominant side and more in women than in men. Thus Todd explained the greater frequency of symptoms of pressure developing in women at the beginning of the adult age.

Much has been written concerning the role played by an aberrant cervical rib in the production of brachial neuritis Such ribs are not common, their occurrence has been between 0.03 and 0.12 per cent in various series of cases studied When present they appear bilaterally in 67 to 80 per cent of all cases. Although the rib is found more commonly on the left, symptoms or pressure are more common on the right In more than 50 per cent no symptoms are produced.[21] The length of aberrant cervical ribs varies from a mere trace to a fully developed rib which joins the sternum anteriorly Intermediate forms commonly join the first rib directly or by fibrous bands at any point along its superior aspect (Fig 11).

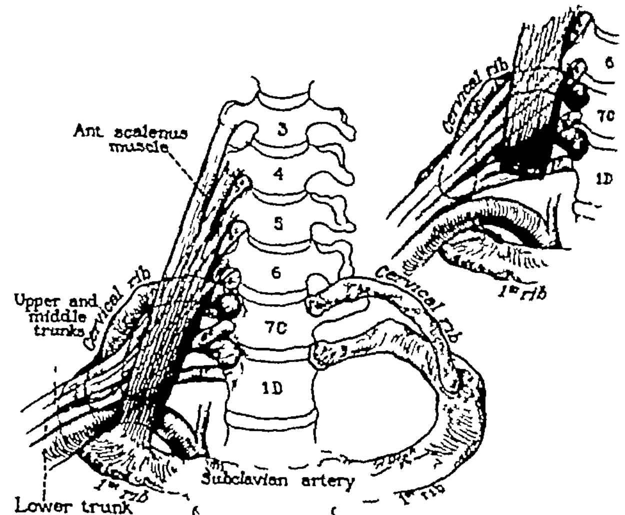


Fig. 11 Compression of brachial plexus and subclavian artery between cervical rib and anterior scalenus muscle (From Love, J G. The Scalenus Anticus Syndrome with and without Cervical Rib. In Allen, E V , Barker, N W , and Hines, E A , Jr. Peripheral Vascular Diseases, 1946, p 309 Reprinted with permission of authors and publisher, W B Saunders Company)

Depending on its length such an aberrant rib may be grooved by the first thoracic nerve alone or by the nerve and subclavian artery. Whether or not such a process will cause irritation of the lower roots of the brachial plexus will depend on several factors, among which are the presence of a postfixed plexus, the position and configuration of the scalenus anterior muscle, and the postural adjustments of the individual Only careful study will permit the roentgenologist to distinguish a cervical rib from a rudimentary first thoracic rib.

The scalenus anterior muscle is closely related to all five spinal roots of the brachial plexus and to the subclavian artery (Fig. 12).

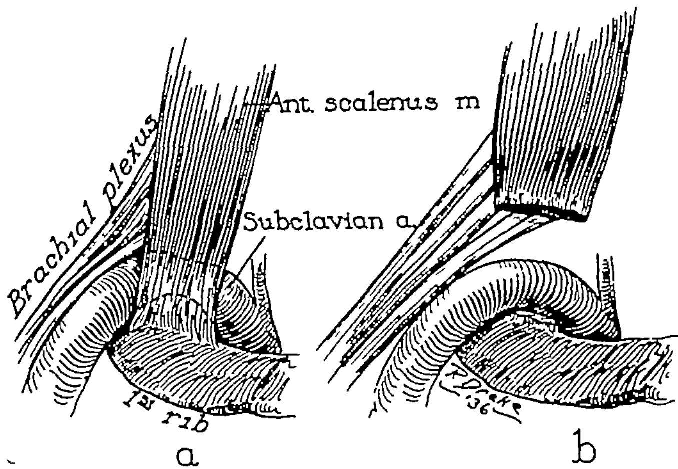


Fig 12 Anatomic relationships illustrating that tension on brachial plexus and subclavian artery may be produced by hypertrophied scalenus muscle (From Craig, W. McK., and Knepper, P A Cervical Rib and the Scalenus Anticus Syndrome Ann Surg 105 556-563 [Apr, 1937] Reprinted with permission of the authors and the publisher, J B Lippincott Company)

The brachial roots may be compressed between the anterior and the middle scalenus muscles or by splitting the anterior muscle in an abnormal fashion[13] (Fig 13) However,

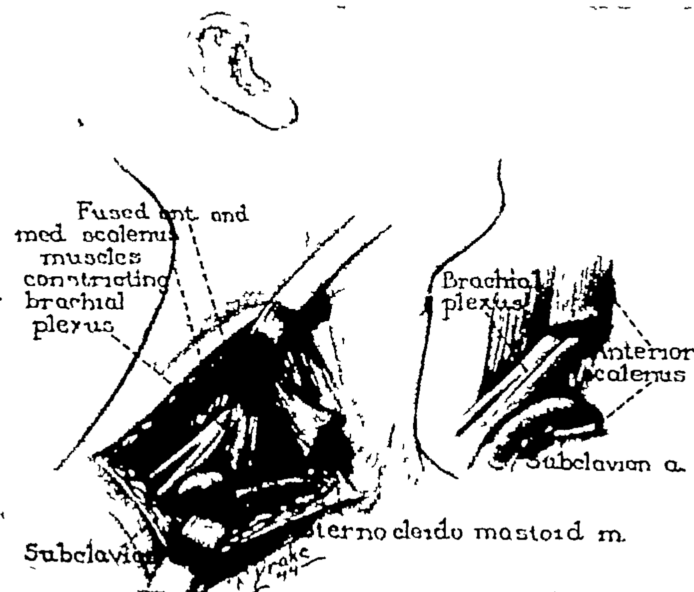


Fig 13 Anomalous constriction of brachial plexus by scalenus muscle (From Love, J G The Scalenus Anticus Syndrome with and without Cervical Rib In Allen, E V, Barker, N W, and Hines, E. A, Jr Peripheral Vascular Diseases, 1946, p 306 Reprinted with permission of authors and publisher, W B Saunders Company)

evidence seems to be increasing that the anterior scalenus muscle per se rarely causes mechanical irritation of the plexus. That it may do so reflexly, from tightening which is secondary to irritation of the plexus, seems likely. Thus irritation of the roots by any of the compression mechanisms mentioned, a herniated disk for instance, might bring about such reflex contraction. In any event, numerous cases are on record in which its section, with or without other operative procedures, gave relief to individuals suffering from a mechanical brachial neuritis. It is interesting to note in this connection, that most of the maneuvers used presumably to test compression by the anterior scalenus muscle, namely, extension of the neck, bracing of the shoulders, deep inspiration, and rotating the head to one or the other side, frequently will increase compression at one or another of the extraspinal sites of predilection now being considered

Falconer and Weddell,[18] in 1943, and Walshe, Jackson and Wyburn-Mason,[19] in 1944, have demonstrated how structures passing from the posterior triangle of the neck into the axilla may be compressed between the first rib and the clavicle (Fig 14). Falconer and Weddell found that approximately 50 per cent of the normal subjects examined showed alteration of the radial pulse on downward and backward bracing of the shoulders. Roentgenographic studies made by these authors revealed an appreciable narrowing of the costoclavicular space during these maneuvers. They advised that when conservation measures fail, removal of a portion of the offending rib be carried out.

Wright,[22] in 1945, described cases in which evidence of plexal and arterial compression appeared on hyperabduction of the arm, especially when long continued as in sleeping in the supine position with the arm extended above the head. As a result of his studies, including dissections, he concluded that, on hyperabduction of the extremity, the axillary vessels and the main cords of the brachial plexus may be pinched between the pectoralis minor muscle and the coracoid process (Fig. 15) He further found that the radial pulse of approximately 80 per cent of 150 normal individuals studied was obliterated by hyperabduction of the arm above the head.

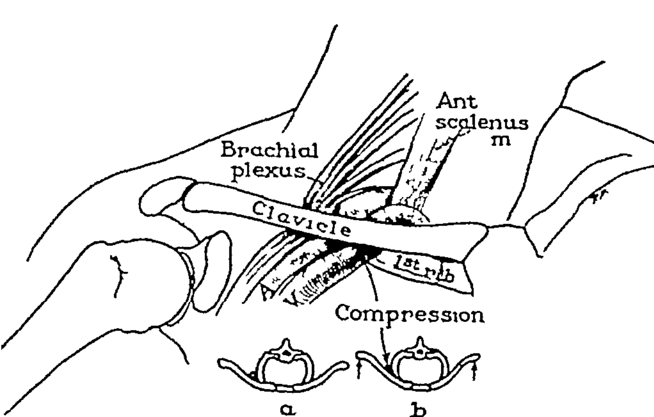
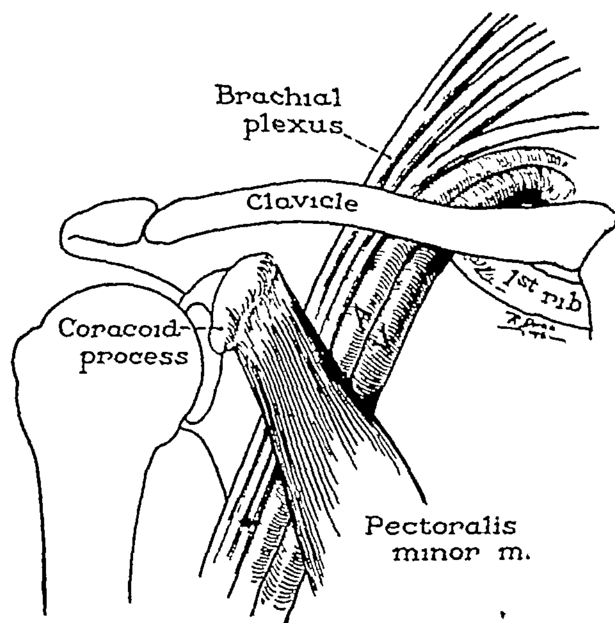


Fig 14 The mechanism of costoclavicular compression described by Falconer and Weddell. (From Eaton, L.M.· Neurologic Causes of Pain in the Upper Extremities, with Particular Reference to Syndromes of Protruded Intervertebral Disk in the Cervical Region and Mechanical Compression of the Brachial Plexus. S Clin North America. 26 810-833 [Aug., 1946] Reprinted with permission of the author and publisher, W.B. Saunders Company)



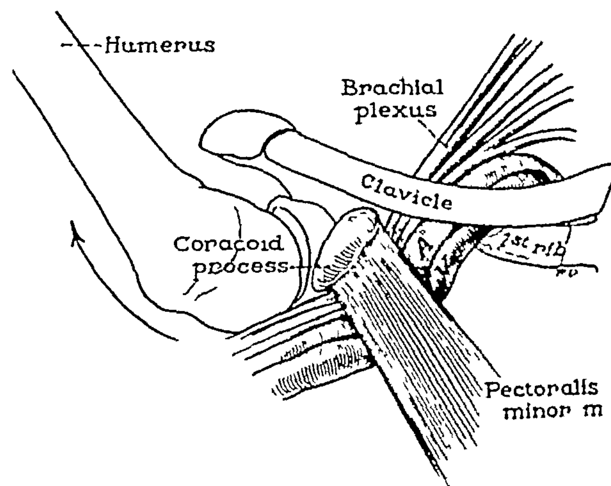


Fig 15. The subcoracoid-pectoralis minor syndrome (modified from Wright's photographs). (From Eaton, L. M Neurologic Causes of Pain in the Upper Extremities, with Particular Reference to Syndromes of Protruded Intervertebral Disk in the Cervical Region and Mechanical Compression of the Brachial Plexus S Clin. North America. 26 810-833 [Aug , 1946] Reprinted with permission of the author and publisher, W. B Saunders Company)

Summary

As the term is generally employed, brachial neuritis includes conditions which more specifically should be designated radiculitis, plexitis, or peripheral neuritis. However, regardless of the term employed, it should be understood to indicate manifestations of disturbance in somatic and visceral functions of the components of the brachial plexus. This review has revealed several possibilities for mechanical involvement of the neural and vascular structures located in the cervical and shoulder regions. Such disorders may produce one or more of the signs of nerve dysfunction, which include somatic sensory, somatic motor, or autonomic manifestations. Clinically one may find pain, paresthesia, objective sensory change, evidence of muscular weakness or atrophy, and cutaneous vascular, or sudomotor disturbances. As a general rule, isolated brachial neuritis may be considered the result of mechanical compression of roots, plexus, or nerves, until proved otherwise.

REFERENCES

1. Eaton, L. M. Neurologic Cause of Pain in the Upper Extremities, with Particular Reference to Syndromes of Protruded Intervertebral Disk in the Cervical Region and Mechanical Compression of the Brachial Plexus. S. Clin. North America. 26 810-833 (Aug., 1946).
2. Brash, J. C., and Jamieson, E. B. Cunningham's Text-Book of Anatomy Ed. 8, New York, Oxford University Press, 1943, 1558 pp.
3. Semmes, R. E., and Murphey, Francis The Syndrome of Unilateral Rupture of the Sixth Cervical Intervertebral Disk, with Compression of the Seventh Cervical Nerve Root, a Report of Four Cases with Symptoms Simulating Coronary Disease. J. A. M. A. 121 1209-1214 (Apr., 1943).
4. Naffziger, H. C., and Boldrey, E. B. Surgery of Spinal Cord. In Bancroft, F. W., and Pilcher, Cobb· Surgical Treatment of the Nervous System. Philadelphia, J. B. Lippincott Company, 1946, sect. 4, chap. 17, pp. 327-406.
5. Eaton, L. M. Pain Caused by Disease Involving the Sensory Nerve Roots (Root Pain), Its Characteristics and Mechanics of Its Production. J. A. M. A. 117 1435-1439 (Oct. 25, 1941).
6. Haymaker, Webb, and Woodhall, Barnes Peripheral Nerve Injuries, Principles of Diagnosis. Ed. 1, Philadelphia, W. B. Saunders Company, 1945, 227 pp.
7. Ray, B. S., Hinsey, J. C., and Geohegan, W. A Observations on Distribution of Sympathetic Nerves to Pupil and Upper Extremity as Determined by Stimulation of Anterior Roots in Man. Ann. Surg. 118 647-655 (Oct., 1943).
8. Swank, R. L., and Simeone, F. A. The Scalenus Anticus Syndrome, Types, Their Characterization, Diagnosis and Treatment. Arch. Neurol. & Psychiat. 51 432-445 (May, 1944).
9. Jones, F. W. Discussion on Cervical Ribs, the Anatomy of Cervical Ribs. Proc. Roy. Soc. Med. 6 95-113 (Feb., 1913).
10. Jones, F. W. Variations of the First Rib, Associated with Changes in the Constitution of the Brachial Plexus. J. Anat. 45 249-255 (Apr., 1911).
11. Todd, T. W. The Descent of the Shoulder after Birth. Anat. Anz. 41 385-397 (June, 1912).
12. Todd, T. W. Posture and the Cervical Rib Syndrome. Ann. Surg. 75 105-109 (Jan., 1922).
13. Adson, A. W., and Coffey, J. R. Cervical Rib, a Method of Anterior Approach for Relief of Symptoms by Division of the Scalenus Anticus. Ann. Surg. 85 839-857 (June, 1927).
14. Craig, W. McK., and Knepper, P. A. Cervical Rib and the Scalenus Anticus Syndrome. Ann. Surg. 105 556-563 (Apr., 1937).
15 Naffziger, H. C., and Grant, W. T. Neuritis of the Brachial Plexus Mechanical in Origin, the Scalenus Syndrome. Surg., Gynec. & Obst. 67· 722-730 (Dec., 1938).
16. Eden, K. C The Vascular Complications of Cervical Ribs and First Thoracic Rib Abnormalities. Brit. J. Surg. 27 111-139 (July, 1939).
17. Hill, R. M. Vascular Anomalies of the Upper Limbs Associated with Cervical Ribs, Report of a Case and Review of the Literature. Brit. J. Surg 27 100-110 (July, 1939).
18. Falconer, M. A., and Weddell, Graham Costoclavicular Compression of the Subclavian Artery and Vein Relation to the Scalenus Anticus Syndrome. Lancet. 2 539-543 (Oct., 1943).
19. Walshe, F. M. R., Jackson, Harvy, and Wyburn-Mason, R. On Some Pressure Effects Associated with Cervical and with Rudimentary and "Normal" First Ribs, and the Factors Entering into Their Causation Brain. 67 141-177 (Sept., 1944).
20. Grant, J C. B. A Method of Anatomy, Descriptive and Deductive. Baltimore, The Williams & Wilkins Company, 1940, 794 pp
21. Woltman, H. W. Neuritis and Other Diseases or Injuries of Peripheral Nerves In Tice, Frederick. Practice of Medicine. (In press.)
22. Wright, I. S. The Neurovascular Syndrome Produced by Hyperabduction of the Arms, the Immediate Changes Produced in 150 Normal Controls, and Effects on Some Persons of Prolonged Hyperabduction of the Arms as in Sleeping, and in Certain Occupations. Am. Heart J. 29 1-19 (Jan., 1945).

THE RELATION OF UPPER EXTREMITY PAIN TO CERVICAL INJURIES

Arthur G Davis, M D

Evidence was submitted at a joint session of the section of Orthopedic Surgery and Radiology, June 1944,* purporting to show the agency of heretofore obscure lesions of the cervical spine as a causative factor in referred pain to the upper extremity. The causative mechanisms, the special reason why these upper extremity lesions have remained unexplained, the misconceptions regarding the use of the word "sprain," and the evidence of what constitutes normal range of motion was submitted. Deviations from such normal ranges were described The concealment factors at work in the cervical spine were discussed A new method of roentgenologic interpretation was presented (Fig 1). Diagnosis of specific obscure lesions was shown to depend upon the correlation of radiologic and neurologic findings Differential diagnosis was based upon a better understanding of the mechanics of production of cervical lesions, special x-ray technique, the fitting together of neurologic and radiologic findings, and the proof of relief attained through hyperextension treatment

Five categories of lesions are differentiated by this approach 1) luxation of the posterior facets, 2) narrowing of the intervertebral space, 3) rupture of the disc, 4) lesions encroaching on the intervertebral foramen, 5) combinations of any two or more Straight-

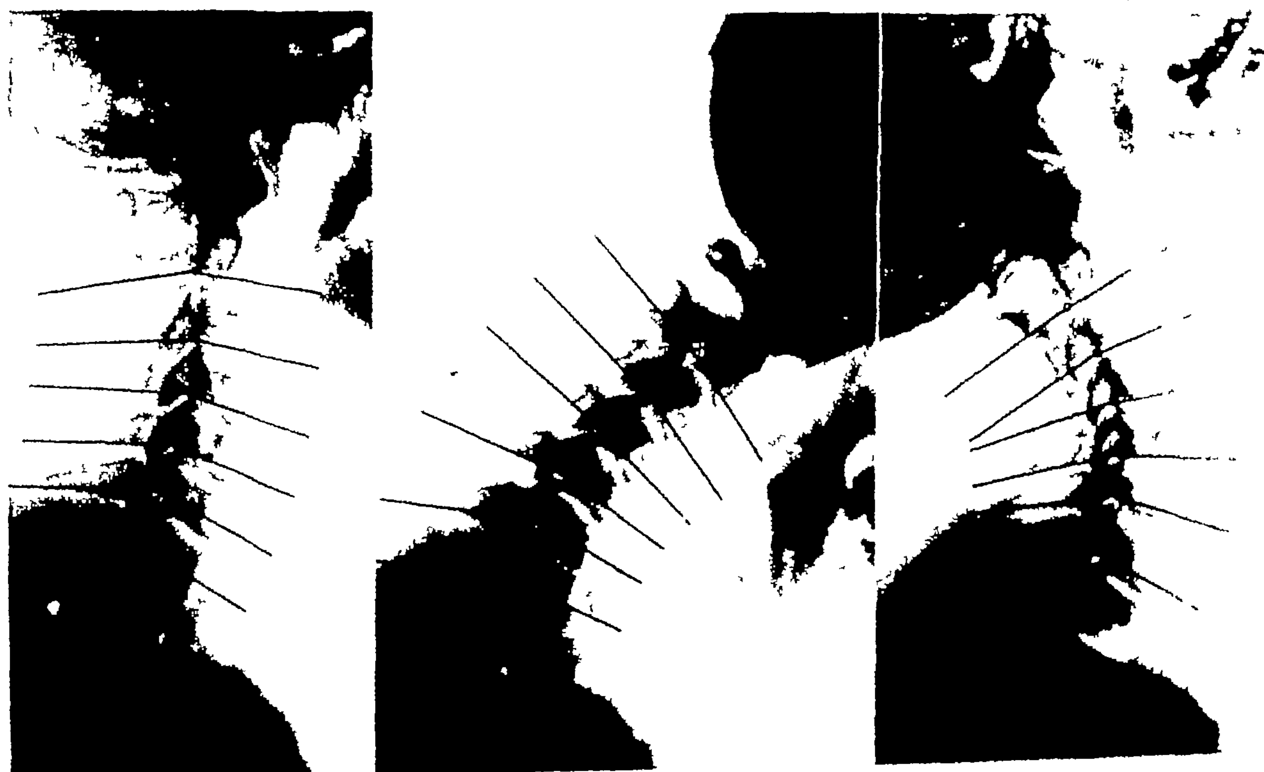


Fig 1 Normal Standard Lines have been drawn to visualize motion to full normal range Left shows sitting relaxed position, eyes horizontal, heavy sand bags (25 lbs) held in each hand to eliminate overcast of shoulder girdle

Center figure shows extreme range of forward flexion (chin chest position)

Figure to right shows extreme of hyperextension

ness was brought out as the most significant and most constant finding indicative of a hidden cervical lesion. This fact, together with the complaint of upper extremity pain, was brought out as a duality of clinical findings sufficient to arouse suspicion and stimulate further investigation of a more definitive nature to establish a specific diagnosis other than neuritis or sprain.

The most frequently overlooked and the most commonly demonstrable lesion was shown to be a partial or complete rupture of the posterior interarticular ligaments Because the original lesion proved relatively symptom-free at the moment of accident or was overlooked by both roentgenologist and orthopedist or treated under the heading of sprain, the true nature of the lesion was missed It was shown that normal use of the head and neck in such neglected lesions resulted in a creeping migration at the point of the torn ligament manifesting itself later as a permanent disability.

In the light of subsequent experience and the accumulation of a considerable number of cases, the following evidence is submitted in support of the contentions set forth in the earlier paper. It is, however, more fitting to approach the subject from the point of view of upper-extremity pain than it is from the point of view of cervical injury. Since most of the cases are ambulatory and come to the physician's office with a complaint of upper-extremity pain of various kinds depending upon the level of the cervical spine involved and the dermatome of referred pain, it is clear that this evidence concerning the cervical spine must constantly be kept in mind in cases complaining of obscure upper extremity pain. Tabulations covering the period 1944 to 1946, inclusive, show one hundred eighty-two such cases analyzed. Of these, according to my diagnosis, ninety-two proved to be luxations, nineteen, disc lesions, eighteen, arthritic, four, spinous process fractures, fifteen, combined disc and luxations, and thirty-four showed no x-ray evidence of an abnormal kind. Of the positive ones, two disc protrusions were operated for the removal of the disc detritus combined with fusion, and two questionable protrusions were fused without laminectomy. The distributions were as follows· Luxations -

C_2 on C_3 . . . 4	C_5 on C_6 . . . 24
C_3 on C_4 . . . 4	C_6 on C_7 . . . 9
C_4 on C_5 . . . 35	C_7 on T_1 . . 2

The distribution of the disc lesions were as follows Disc -

C_4 & C_5 . . . 2	C_6 & C_7 . . . 5
C_5 & C_6 . . . 10	C_5 & C_6 and C_6 & C_7 . . 2

There were fifteen combined disc and luxation lesions occurring as follows

Luxation C_4 on C_5	Disc between 5 & 6 . . . 6
Luxation C_5 on C_6	Disc between 6 & 7 . . . 3
Luxation C_4 on C_5	Disc between 6 & 7 . . . 2
Luxation C_3 on C_4	Disc between 4 & 5 . . . 2

There was one case in which there was a double luxation between C_4 and C_5, C_5 and C_6 and a disc lesion between C_4 and C_5 and C_5 and C_6. Two cases of scalene syndrome were identified, one of whom had a scalenotomy, which relieved the symptoms partly but later required a fusion to eliminate a recurrence of painful symptoms. The other also showed roentgenologic evidence of luxation and disc involvement, but the scalene syndrome seemed to be the outstanding feature. This case was treated conservatively and so far has not required operative interference.

The commonest complaint was of unilateral shoulder pain Common complaints in the order of ther frequency are as follows· bilateral shoulder radiation, whole-arm radiation, radiation to the radial side of the hand, suprascapular radiation, lateral side of the neck, suboccipital, and precordial, together with various combinations of the above.

It is becoming more than ever evident from observation in the one hundred eighty-two cases herewith analyzed that the simple procedure of comparing the two lateral x-ray projections, one relaxed, the other flexed, is the most revealing (Figs 1, 2). While other views of spine may be important, they are considered distinctly secondary to the lateral projection in these two positions The standard anteroposterior is necessary for detection of cervical rib and transverse process fracture. Bilateral oblique films are obviously necessary to determine aberration of the intervertebral foramina and fractures of the articular processes, or anomalies and arthritis. Lamina-

grams and myelography may be resorted to, but they are secondary.

A careful history on the precise area distribution of pain, paresthesia, hypesthesia, or hyperesthesia is important (Fig. 3). The areas most frequently encountered are as follows shoulder, whole arm, radial side of the hand, ulnar side of the hand, particular finger, suprascapular, suboccipital, lateral cervical, and precordial.

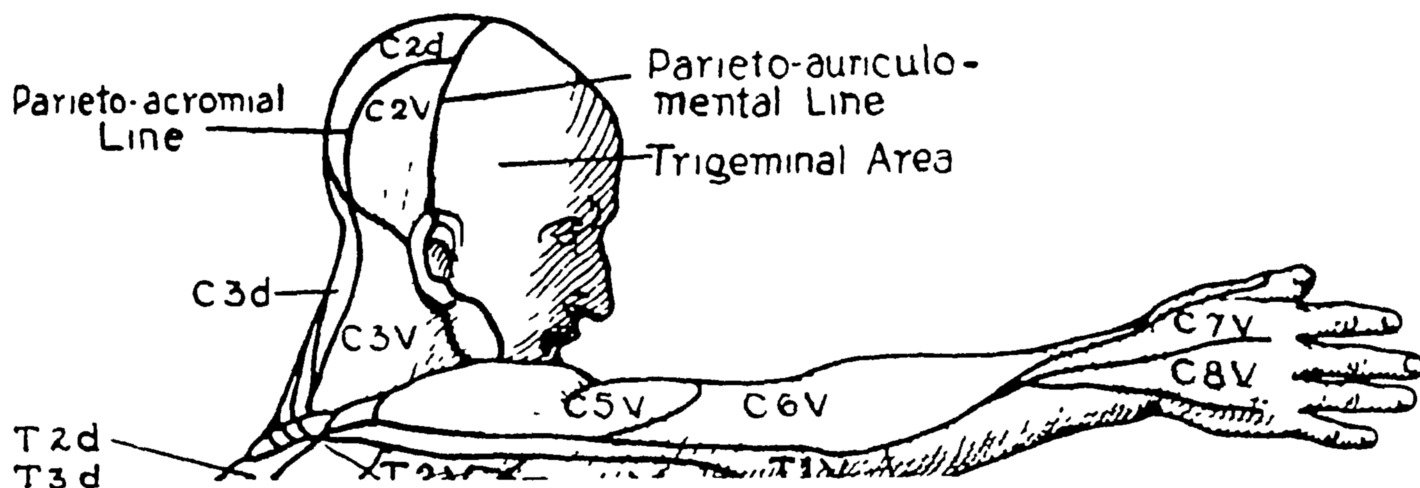


Fig 3 Outline of areas commonly involved in cervical spine lesions From Tilney, Frederick, and Riley, H A Form and Functions of the Central Nervous System, ed 2, figure 174, page 166, the somatic dermatomes (Dejerine), courtesy of Paul B Hoeber, Inc , New York

The differential diagnosis between actual pressure on nerve roots, as in disc lesions, or pain radiation, as in the luxation type, depends upon whether or not the segmental areas just mentioned show actual organic change. If by comparison with the normal side, the reflexes can be demonstrated to be diminished or absent and there is actual regional atrophy, such as triceps, biceps, or interosseous, differentiation is made easy. Jugular compression, coughing, sneezing, and vertical compression of the head tilted to the affected side increase pain in ruptured disc lesions. Differentiation by myelography may be indicated. Further differentiation with the scalene syndrome in mind indicates the necessity of determining temperature sense, swelling, discoloration, difference in the pulse volume, comparative blood pressure readings, pressure over the scalenus anticus attachment, and resort to the capillary thermocouple.

Regarding roentgenologic interpretation the following special points of observation seem indicated particularly in the light of therapeutic proof based on observations made in this manner. The following points in the order of their importance have served to reveal otherwise obscure lesions straightness of the cervical spine in the lateral relaxed projection (Fig. 4), deviation from the normal symmetry of forward curvature in the forward flexed position; forward displacement of body on body (Fig 5), and deviation from the parallel of the posterior facets. In addition to the above, chip centrum, chip fractures of the tip of the spinous process, old ununited fractures of the spinous processes, and beading of the posterior borders of the centra are also common observations serving to indicate the level of the lesion. Apparent fusion of the intervertebral space, irregularities of the articular surfaces of the articular processes, and comparison of the posterior borders of the centra as indicated in Fig 2, and lastly in the antero-posterior view notation of old fractures of the transverse processes and the question of cervical ribs constitute the other points to be noted

If the roentgenologic and neurologic findings coincide and if the dermatome of neurologic signs corresponds exactly with the level



Fig. 4. Typical Well-Defined Luxation C_4 on C_5. The left figure shows typical straightness in the sitting relaxed position

Right figure shows sharp deviation from symmetrical forward curve between four and five. Also note unusual spread of the spinous processes at this point

Patient thrown by the explosion of the gas tank six months prior to the first observation Pain radiation to both shoulders No cervical symptoms Shoulder pain disappeared immediately upon application of plaster collar in hyperextension Collar worn three months No recurrence so far one year later

of x-ray change in the cervical spine, the cause is ordinarily extablished. The problem then resolves itself into the question as to whether there is nerve-root compression with permanent signs such as atrophy, reflex change, and hypesthesia, such as is present in disc protrusions, or whether the pain is of the referred nature without such permanent changes but covering the same segmental area as if actual root or plexus pressure did exist. Cases presenting the referred type of pain are of the luxation type. Both types, whether discs or luxations, must be differentiated from the scalene syndrome. There have been a number of cases presenting upper extremity symptoms of an obscure nature in which no lesion of the extremity itself could be demonstrated and the cervical spine exhibited a departure of one kind or another from the symmetrical arc of curvature in the forward flexed position, but the evidence was slight, rendering a radiologic decision as between normal and abnormal im-

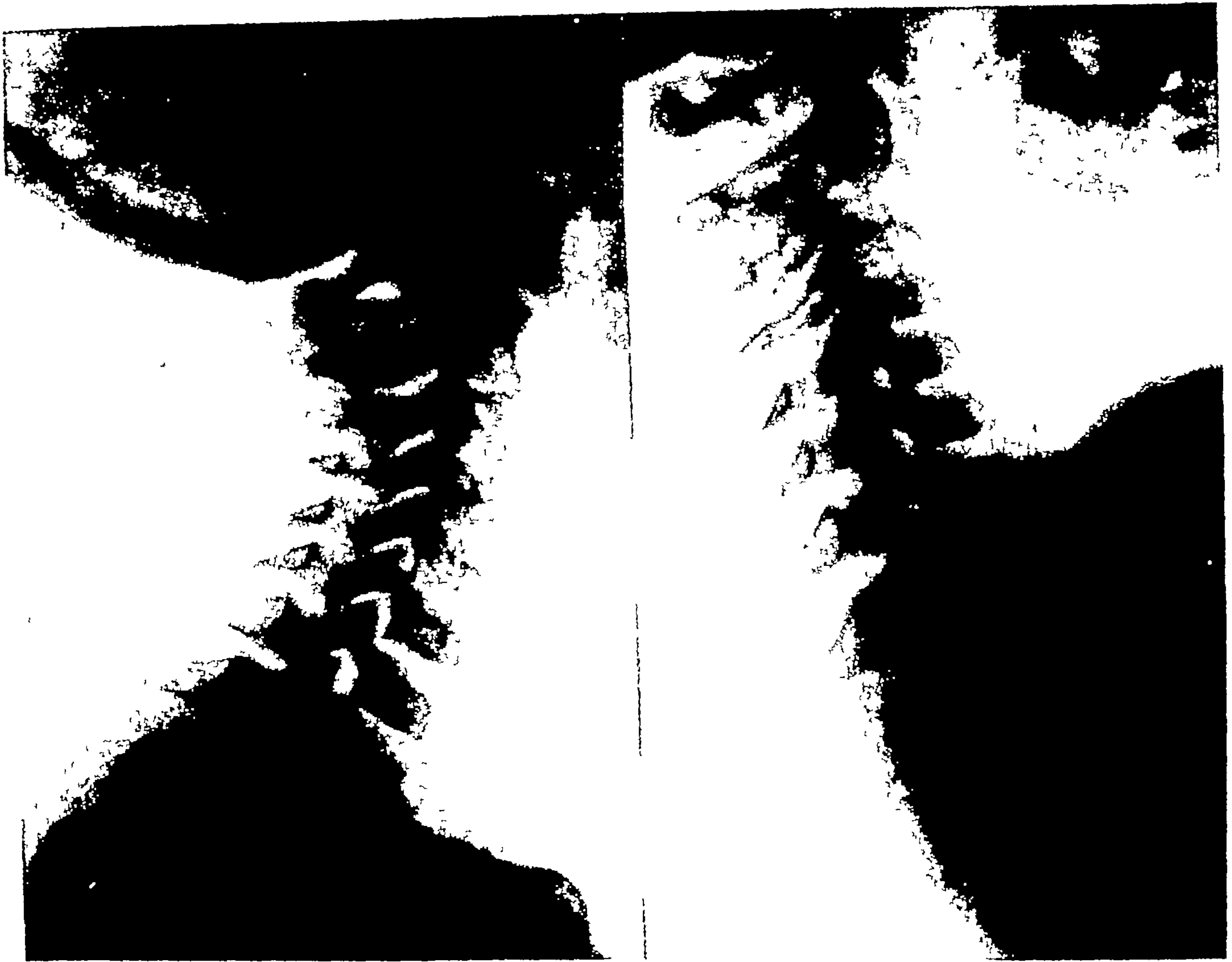


Fig 5 Luxations C_2 forward on C_3. Automobile accident - seven-year-old boy. Was seen within a few hours and each time he attempted to sit up he became dizzy and fell over Complaint of pain in upper cervical spine.

Note the left figure shows no deviation from the normal forward curvature. Note on the right the projection of the 2nd forward on the 3rd.

Hyperextension treatment in a plaster collar permitted the child to be up and about the following day Collar was worn one month - complete recovery

Note this luxation undoubtedly would have escaped diagnosis had it not been for the lateral projection in the forward flexed position.

possible. In a number of such cases the pain was relieved by the application of a cervical collar in hyperextension. In the more outspoken cases of luxations the hyperextension collar results almost universally and in a very short time, a few hours to a day, in complete relief of upper extremity symptoms. Thus, a test of therapy based on agreement between roentgenologic and neurologic findings usually provides conclusive evidence of the causative mechanism at work in most obscure cervical lesions.

The treatment of the various type cases contained in this group is as follows for recent luxations a hyperextension collar is applied for three to four weeks, for old luxations, up to three months, depending upon the degree and time interval since accident. For the combined lesions, where the disc and luxation features are both present, the same rule holds. Recent lesions uncomplicated by fracture as a rule recover completely with no threat of recurrence after four weeks of such hyperextension. The patient is up and about throughout

treatment but unable to work because of the extreme position of the head. Quite a number of the old lesions have recurred following the removal of the collar (Fig. 6). In such cases collars are again applied and if a second trial has the same results, operative fusion of the vertebrae is considered (Fig. 7). Each case is ordinarily given postural exercises following the removal of the collar. Several cases have undergone a three-vertebrae fusion after one



Fig. 6 Gross Dislocation. This case is shown as a demonstration of the complete avulsion of all posterior ligamentous structures. The left figure shows the sitting relaxed position of the patient when first seen one month following a fall down a flight of steps The center figure shows exaggeration of the dislocation in the flexed position The right figure shows the patient after three months of treatment in hyperextension

In this case there was pain radiation down both arms for several days The pain cleared up spontaneously No medical attention When first seen she walked into the hospital with slight arm and cervical spine symptoms After three months of hyperextension treatment, luxation occurred to a lesser degree, fusion was recommended Patient declined further treatment

Note comparison with the other figures shows this case to be an extreme degree of the same mechanism of whip-lash experienced in most cervical injuries In this case it seems clear that the capsule of the posterior articular processes is completely avulsed, the posterior common ligament and probably partial avulsion of the annulus fibrosis posteriorly with disruption of the entire intervertebral disc Healing by scar of the torn ligaments during the 'golden period' of the first three weeks accounts for the failure, as it also accounts for the creeping migration of cases of lesser degree



Fig 7. Combined Luxation and Disc Involvement When first seen patient complained of right arm symptoms, swelling, weakness, and pain to the radial side of the hand and the index Was first referred neurosurgical Scalenotomy was done with relief of most of the symptoms Recurrence of painful symptoms three years later. Referred Orthopedics - history of head-on automobile collision fourteen years prior

Note on the left straightness of the cervical spine and on the right narrowing of the intervertebral space between the C_5 and C_6 and luxation of the 4th forward on the 5th

Hyperextension collar relieved symptoms immediately. Wore the collar three months in which there were no symptoms in the right arm Recurrence shortly after collar was removed. Fused 4th to 6th Convalescence in hyperextension again three months No recurrence of symptoms during the last 18 months

or two such recurrences with apparently good and permanent results. Cases showing conclusive evidence of disc rupture with organic change in the segment involved are laminectomized and fused at the same sitting (Fig. 8). So far every case showing organic change from disc protrusion has also shown the luxation feature, either at or above the level of the disc damage (Fig 9, A and B) From this observation it would seem therefore that, if one stopped at the point of simply removing the disc detritus and relieving the pressure on the nerve root, the symptoms in the same general area would persist because of the luxation. End-result studies in this series of cases are not significant because they are too recent. An end-result study is being started, it will cover all groups and will be in terms of three years.

Cervical injuries sustained in street accidents, especially head-on collisions, form an-



Fig 8 Combined Disc and Luxation Lesion. Note on left, straightness of cervical spine
Center, narrowing of intervertebral space between 5 and 6, luxation forward of 4 and 5, small shadow anterior to upper forward angle of the 5th
Right figure shows fusion following disc and fusion operation.
When first seen patient was referred to the hospital and given opiates for right arm pain. Pain extended down to the thumb and index finger Halter traction relieved symptoms Jugular compression caused exacerbation of sharp pain in the right extremity Vertical compression of the head bent to the right side also increased pain Combined disc and fusion operation, hemilaminectomy of the sixth cervical Loose material immediately presented extradurally Result slight limitation of motions of the head and neck, otherwise no recurrence of disability for last year and a half
History- Patient was injured in a head-on automobile collision fourteen months prior to first being seen No x-ray was taken of the cervical spine at that time because of absence of symptoms. Patient did not recall pain down the arm at that time. Several times noted slight pain down the arm at long intervals. Finally while playing golf, sudden onset of excruciating pain down the right arm. X-ray of the cervical spine was not suggested until he finally entered the hospital completely disabled
Fusion extended 4th to 7th because of luxation of the 4th

other group of cases which require mention at this point. Medical responsibility begins at the site of the accident. Ambulance attendants should be instructed, in case of cervical injury, to place the head in hyperextension with a blanket roll under the neck, an attendant should remain at the head of the patient to maintain this position. On entering the hospital the patient should immediately be put to bed and the head transferred from the hands of the attendant to a halter with five pounds of traction, the head of the bed should be elevated. At this point, but not before, should the x-ray survey take place. The experience of others as well as our own has shown that patients who showed no paralysis at the time of accident have developed neurologic signs enroute to the hospital or as a result of improper handling of the head after the patient arrives at the hospital.

Gross fractures and dislocations are immediately identified by the survey x-ray. If the survey proves negative for a gross lesion, it does not mean, however, that the patient is negative for the obscure variety of injury It is our practice, therefore, that after the pa-

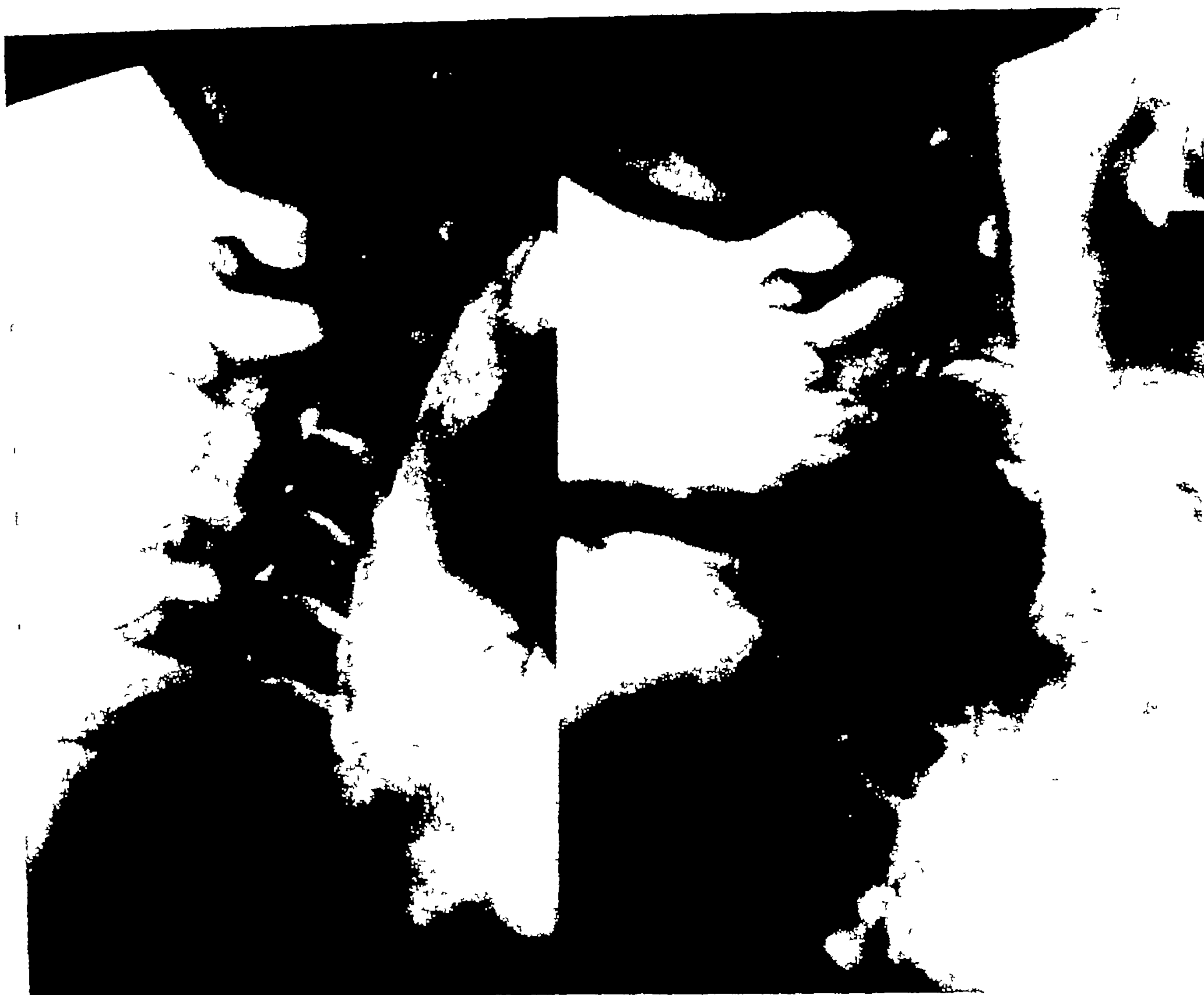


Fig. 9A Combined Luxation and Disc Lesion C_6 on C_7 Patient sustained a fall down several steps one year before observation Complaint - pain, atrophy, and weakness of left upper extremity Triceps markedly atrophied Triceps reflex obliterated Full arm pain but extending to the index finger and thumb.

Left figure shows marked spreading in the forward flexed position between the spinous processes of the 6th and 7th, also note marked narrowing of the intervertebral space and traumatic arthritis

tient is up and about, a second series of pictures is taken in the sitting relaxed position and in the chin-chest position Such a second series will frequently disclose the luxation type of injury. The opportunity thus presents itself to provide proper treatment with a hyperextension collar to permit healing of the torn ligaments in the short position during the 'golden period' of the first three weeks when fibrous union takes place. If these lesser lesions are left untreated the majority will heal by scar, and the normal use of the head and neck will induce a creeping migration of the vertebra involved, producing the luxation syndrome frequently encountered from months to years later. It is the author's opinion that, in spite of the absence of concrete proof, injuries of the intervertebral discs occur in the same manner and as a result of the same mechanical force (whip lash) and whenever the posterior capsular ligaments are torn, the posterior fibers of the annulus fibrosis are also torn. The evidence of this is to be seen long after accident, when the telltale marks that are left show as narrowing of the disc, complete obliteration of the disc, or beading of the poste-



Fig 9B. Shows the two oblique elevations and the Pantopaque notch Hemilaminectomy on the left side of the sixth revealed no loose material but an impingement of an exostosis on the nerve root was found

A fusion was done between the fifth and seventh vertebrae A lesser degree of symptoms persisted in the left arm during the first month of convalescence Patient has just been released after three months of hyperextension collar but has been back at some of his duties as school teacher during the last month.

rior borders of the two vertebrae adjacent to the involved disc. Besides, it is a known fact that disc and luxation lesions are frequently associated at different levels in the same case.

At this point it seems appropriate to repeat the warning of the extraordinary concealment factors involved in cervical injuries The screens which have hidden the real facts are of several kinds. The first deception is as to the magnitude of the lesion. Whether the injury be due to a car accident, a headlong fall, fall from a height, slipping on a greasy floor, letting go while pulling on long-handled wrenches, or various kinds of athletics, it is produced in the great majority of cases by the thrust forward and downward of the weight of the head beyond the range of normal flexion. In the instant following, the automatic recoil tends in part to reduce the magnitude of the injury, whether it be fracture, dislocation, or luxation, so that by the time the first x-ray survey is made a degree of reduction has taken place in almost all cases except those in which the processes have jumped and locked. Meanwhile, regardless of the normal or near normal appearance in the first x-ray, any or all of the posterior ligaments may have partly or completely avulsed. The automatic recoil mechanism is dual in character. Binocular vision demands the head to be centered vertically and the eyes to be horizontal The first instinctive reaction, therefore, is to recover this equilibrium. The strong posterior static musculature, the ligamentum nuchae, and the flava ligaments constitute the other automatic mechanism initiating the recoil from hyperflexion. Thus it will be seen that the extent of the injury at the moment of accident is never known, but because of these self-evident recoil factors the extent must be allowed for. It is clear, therefore, that the difference between the x-ray taken at the hospital and one taken at the moment of accident, if this were possible, would be very considerable.

Regarding the ambulatory patient who comes to the office complaining of upper-extremity pain, another kind of concealment factor is introduced. If he has sustained a partial or complete avulsion of the posterior capsular ligament, he tends automatically to lean away from these injured joints. He pulls in his chin, thus straightening the cervical curvature, and this becomes habitual with him. This elimination of the forward curvature of the cervical spine is recorded in the first lateral projection taken

in the sitting relaxed position and provides the clue in many of the cases here cited for further study toward the point of a definite diagnosis. It must also be realized that, contrary to ligamentous tears in more superficial regions like the ankle joint and knee joint, there is no evidence of ecchymosis, hematoma, swelling, or tenderness because of the dense, deep fascia containing the crowded structures of the neck and because of the great depth of the lesion (several inches). As is true elsewhere in the spine, roentgenography is at a great disadvantage in exhibiting lesions of the posterior arch. No less than seven dense cortical structures tend to obscure each other in the lateral plane, and no less than five cortical layers overcast each other in the antero-posterior plane. It must be assumed therefore, that probably only some of the lines of fracture show in the usual survey pictures. Resort to special technique characteristic of modern roentgenography show more aberrations from the normal. Nevertheless it must be assumed in the beginning that roentgenograms, however well taken, cannot be expected to show all the details of a particular injury. In most cases a correct evaluation can be reached only scanning the x-ray plate with this fore-knowledge of its limitations and by diagnosing by inference from the positive findings plus the neurologic evidence

After having gone through the experience of Minerva jackets, cuirasses, braces, and various types of collars, the collar shown in Fig. 10, A and B, was evolved in order to

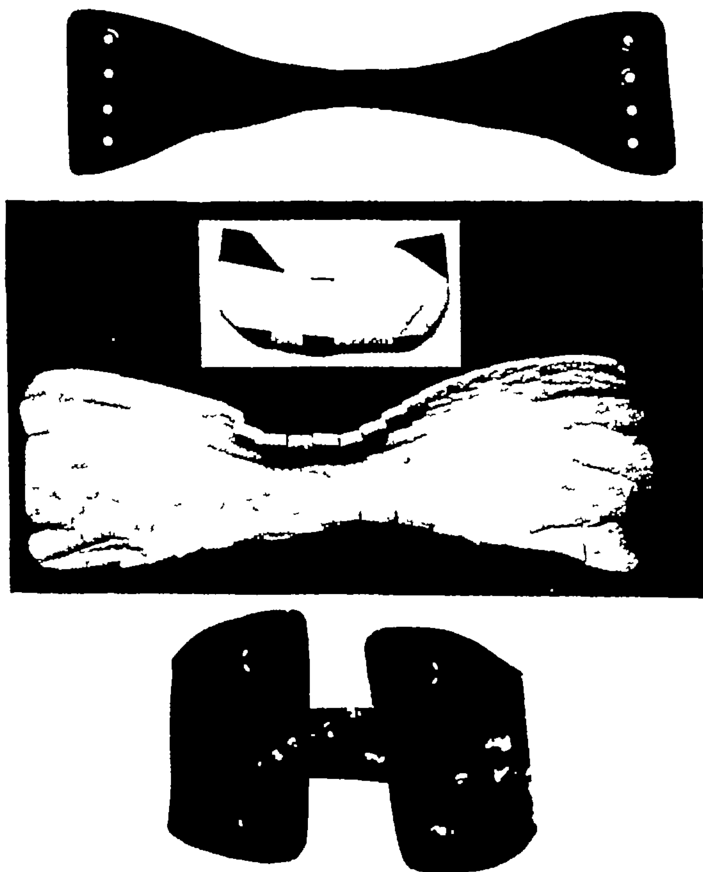


Fig. 10A



Fig. 10B.

Fig 10. Detail of Hyperextension Collar A piece of felt as shown is cut long enough in front to overlap the mandible, fit the contours of the neck and overlap the clavicle. Upper and lower edges are cut to permit a rolled edge The felt is covered with a tube of stockinette, fitted around the neck and then a piece of thin rigid board (paper-thin insulating fabric) is cut, flared at both ends and cut away to a very narrow center portion behind This cardboard is fitted around the felt, tied together in front, a few layers of sheet wadding covers the cardboard and a plaster bandage is made to enclose the collar so that it is irremovable While the plaster is hardening, firm pressure is made on either side of the midline in front and underneath the mandible. In some cases where extreme hyperextension is required, the entire center section behind may be cut away because of pressure on the nape of the neck Additional felt is put under the collar assembly as indicated in Fig 10B

maintain an adequate degree of hyperextension comfortably whether the patient is convalescent, in bed, or ambulatory. Most of the apparatus devised to maintain hyperextension has failed because of inadequate separation of the space between the chin and the chest The inevitable tendency of the patient is to attempt to squirm out of most apparatus in order to talk and eat comfortably Most braces do not provide sufficient distribution of surface bearing to be comfortable. They also have the disadvantage of presenting too much obstruction behind to allow the backward tilting of the head It seems unnecessarily rough to include the entire torso as well as the head and neck for lesions of the cervical spine. The collar assembly shown seems to meet all the requirements of convalescence whether the case is one of serious fracture dislocation or a lesion of lesser magnitude It also possesses the advantage of being irremovable and therefore foolproof. The collar can be jigged up as necessary by adding additional layers of felt under the bottom. It is important to reduce the total substance of felt, cardboard, and plaster to the absolute minimum posteriorly, otherwise full hyperextension will not be realized.

Conclusion

From the facts already in hand the following conclusions seem justified

1 Luxations of the cervical spine are of very frequent occurrence For one reason or another the recent luxation has been overlooked or neglected Luxations are identified as such only months or years after the accident Special x-ray technique is necessary to identify the lesion.

2 Disc protrusions can in most cases be differentiated from the luxation, though they frequently occur together and from the same cause.

3. Hyperextension is by far the most important therapeutic measure for cervical lesions regardless of the degree of severity.

4 Fusions should accompany most lesions primarily operated for disc protrusions

5 The immediate results of therapy as outlined are of sufficient value to justify the observation that hitherto unexplained upper-extremity pain is very frequently due to both recent and old accidents to the cervical spine

BRACHIALGIA

Carl E. Badgley, M.D

The recent advances in the treatment of sciatica have doubtless stimulated both patients and doctors to a renewed interest into a similar situation in the upper extremity termed by Nachlas, "Brachialgia."

Pain radiating into the shoulder, arm, and forearm, so-called brachialgia, presents a symptom complex produced by many various etiological factors. Pain radiating into the arm is the common factor in this syndrome.

Nachlas has emphasized that the symptomatology in this group was essentially due to sensory irritation. Stimulation of sensory areas ranging extensively from the cortex to the periphery, to the distal end of the nerves The irritating stimulus might be 1) within the skull, 2) occipital cervical junction, 3) within the cervical spinal canal, 4) in the intervertebral foramen, 5) supraclavicular region, 6) subclavicular area, 7) the shoulder, and 8) the thorax Clinically, Nachlas has pointed out common lesions with which the syndrome is associated. It is important to recognize that the sensory irritation is not necessarily of central nervous system origin, but may be of muscle, skeletal, or tendinous origin

There are, however, common causes for mild brachialgia of short duration such as is commonly seen following respiratory infections or focal infections, usually producing a myositis of the posterior cervical muscles with radiation of pain into the arm

There were several reports recently in various theaters of war in the development of pain and discomfort about the shoulder girdle followed by paralysis chiefly involving the ser-

ratus anticus, the deltoid, and the supra and infra spinatus muscles, essentially appearing as an acute infectious lesion. In my own practice, there has been an increasing incidence in serratus anticus palsy within the past two years, which may mean that these isolated palsies may develop more commonly then we think.

It is recognized that there is a relatively frequent association of localized palsy of a muscle such as the serratus anticus after receiving horse serum therapy. It seems reasonable to suppose that some type of toxic neuritis has developed in these cases.

These are the more common general causes of brachialgia which are more or less readily interrupted or never reach the doctor as they get well so quickly.

Vitamin deficiency produced by improper diet and frequently alcoholism is a definite factor in the occasional case. One must always be on guard to analyze each case of brachialgia and not jump to conclusions from a preconceived idea or from pet concepts As an example there follows a brief case report on a very good friend of mine, known to be an alcoholic of years' duration, who consulted me with the serious picture seen not infrequently in this group of cases· the right hand was definitely swollen and edematous. There was a history of pain in the shoulder and arm associated with numbness and tingling into the fingers, no definite segmental involvement. There was definite motor weakness, grip weak and extensors weak There was no limitation of shoulder motion, but pain in the external epicondyle and right shoulder particularly.

Inquiry into his drinking and eating habits disclosed that he ate practically no breakfast or lunch, drank about eight gin cocktails during the day, ate a fair dinner, and had several whiskey highballs in the evening.

I referred him to the Department of Metabolism for vitamin deficiency on an alcoholic basis. They found no evidence of vitamin deficiency and suggested that x-rays of the shoulder and elbow should be taken to rule out calcium deposits. To my chagrin, the calcification appeared in both the supraspinatus tendon and in the radio-humeral bursa. Roentgen therapy gave almost immediate relief.

The differential diagnosis of brachialgia is not always easy. One must regard pain in the shoulders and arms as merely a symptom akin to abdominal pain or sciatica. There are multiple factors which can produce the typical radiating pain of brachialgia. Diagnostic acumen and all the scientific aids possible are usually required to diagnose the lesion successfully.

We have found it helpful to immediately divide the cases of brachialgia into two groups, 1) neurogenic or of central nervous system origin and 2) non-neurogenic or without definite evidence of central nervous system origin. The neurogenic type must present definite objective evidence of organic nerve change. The non-neurogenic must show no true objective evidence of nerve involvement.

The neurogenic changes which we look for are segmental sensory changes, motor weakness, reflex changes (recognizing C-5 and 6 supply to the biceps), and fibrillation of muscle fasciculi, pain usually of two types, one of radicular nature - true sharp severe pain momentary in type radiating the full extent of the involved nerve - the other a more or less dull aching constant pain, numbness in fingers associated with tingling may be on a vascular basis but may also have a neurogenic origin.

The non-neurogenic case will show no true sensory, motor, or reflex change. There should be no fibrillation. Atrophy may be present from disuse. Pain will be of the dull aching character and not of the radicular or thalamic type. Numbness and tingling may be present and may be on a mechanical vascular basis.

The importance of the division into neurogenic and non-neurogenic brachialgias is just as great as a similar division in cases of sciatic pain. In our experience, fifteen per cent of our sciatic patients have neurogenic signs, eighty-five per cent non-neurogenic. The neurogenic type may require surgery for improvement, the non-neurogenic usually responds under conservative care.

Pain cannot be utilized as the diagnostic factor in brachialgia. Pain in the arm is common to all cases. The type of pain, however, can often give a definite aid. We are all aware of the shoulder and arm pain associated with the precordial pains of cardiac origin. It is less commonly known that stimulation of the diaphragm produces pain in the tip of the shoulder. This is recognized as visceral reflex pain and is an accepted clinical fact.

Clinicians, however, have not entirely accepted a concept of reflex pain of somatic or-

igin, such as the shoulder joint, instead of pain of visceral origin or directly connected with the central nervous system. It is, however, clinically recognized that the joint pain from a hip lesion is felt by the patient not only in the groin but frequently in the knee, a referred pain of somatic origin

Steindler has clinically utilized a method of differentiating pain referred from a peripheral irritant area, the so-called posterior syndrome, from pain of central nervous system origin by using a novocaine block in the trigger zone of pain He has postulated 1) if the needle reproduces the symptoms on entrance in the trigger zone, and 2) novocaine injection reduces the pain, it is evidence of the peripheral nature of the irritation and excludes spinal origin nerve root irritation

I am doubtful of the academic accuracy of this conclusion, clinically, however, it seems to be of value.

One of the difficulties in localizing the lesion of neurogenic origin producing brachialgia is the loss of definite segmental innervation of the limbs by the construction of the plexuses. Various observations, Sherrington, Head, Foerster, have attempted to unravel the segmental sensory innervation. It was Head who used the term dermatome; Sherrington called it a skin area. The construction of the brachial plexus makes it difficult to have segmentation without overlapping of nerve roots since it is one of the purposes of the plexus to see that no single nerve root supplies any one part.

It recent years (1942), Kellgren and Lewis, by injection of a five per cent saline solution into the rhomboid area of patients, have produced radiation of pain peripherally to the shoulder and arm along the fifth cervical nerve root motor supply. Inman and Saunders, later applying mechanical and chemical irritation to the tendinous structures, the periosteum, and bone on normal human subjects, were able to produce characteristic pathways for pain, which, however, did not correspond to the dermatome. They applied the term scleratome to designate this deep radiation, although embryologically there is no scleratome segmental formation known in the extremity

I believe we can return to Sherrington's work published in 1894, in which he demonstrated conclusively that the motor nerves carry one-third to one-half sensory nerves to the muscles It has been recognized for years that in the early development of the limb bud the nerves grow into the limb bud, and, as the limb bud grows outward, it carries the nerves composing the central parts of the plexus distally the full length of the extremity with the upper and lower portions of the plexus supplying the cutaneous portion of the limb bud and the central portion being practically devoid of cutaneous innervation except at the fingers In fact, there is some question whether the seventh cervical supplies any cutaneous area On the other hand, these centrally placed nerves have sensory fibers carried through the motor nerves which supply the muscles and periosteal tissue deep in the limb bud and at a much higher level than the dermatome. In the leg, for example, the fifth lumbar and the first sacral nerves cutaneously supply only below the knee but supply the gluteals and hamstring muscles In sciatica the patient has severe pain deep in the buttock and thigh and may have radiation into the dermatome area This cannot be explained in the dermatome concept, but it can be explained on the basis of the knowledge of the nerve root motor supply which carries the sensory nerves deep into the structure Preferably, however, is the term emphasized by Sherrington, "skin field and root field."

The significance of this statement may not be understood. The attempt neurologically to localize the cause for pain by segmental, cutaneous, and dermatome alterations is unsound to my mind. The deep muscle soreness and the areas of tenderness are important and there is every evidence that their location is segmental, skin field and root fields should be sought

Tenderness has usually been accepted as an area in which pain can be elicited by pressure, denoting underlying pathology. I believe that tenderness is deep pain in the sensory nerves of the non-cutaneous motor nerves supplying the muscle, periosteum, and tendinous structures and that it is not necessarily of localizing importance. Inman and Lewis have demonstrated and produced the radiation of pain into this area. It is clear from the schematic drawings of Dejerine comparing dermatome and scleratome innervation, that the dermatome is restricted to the muscle fascia and bone, in comparison with the extensive and continuous innervation (scleratome).

The muscle fascia and periosteum innervated by any one root forms a continuous series extending from the trunk directly into the extremity. The dermatomes are isolated areas overlaying only a portion of the corresponding dermatome.

I feel that it would be less confusing if the terms "root field" and "skin field" were employed.

The etiological factors producing brachialgia on both neurogenic and non-neurogenic basis may be divided, according to Nachlas, into zones or regions. A somewhat more adaptable method in considering the neurological aspects is to utilize McNaughton's grouping

1) Lesions localized within the spinal cord and canal,
2) Lesions associated with disease of the vertebral column,
3) Lesions outside the vertebra.

We will follow this classification.

(1) Neoplasms of the cord may produce pain in the shoulder and arm. The character of the neurological changes, of course, is dependent upon the location and size of the lesion. If the nerve root is involved, the pain will tend to be of the nocturnal type, with segmental motor and sensory changes and reflex as well, associated with atrophy. If there is pressure on the cord, we will find tract changes such as pyramidal tract with spastic muscles, clonus, Babinski reaction. If the spino-thalamic tract is involved, there is apt to be a bizarre ill-defined pain associated with loss of sense of position of the extremity and loss of temperature sense. A Brown-Sequard syndrome may develop or a syringomyelia. Spinal puncture is indicated with such changes and fluid alterations are helpful in the diagnosis. X-ray may show widening of the pedicle or alteration in the spinal foramina

(2) Platybasia. In recent years there has been a growing clinical concept of neurological syndromes developing in cases demonstrating congenital abnormalities of the occiput and cervical spine. The short neck and low hair line posteriorly associated with congenital malformation of the cervical spine designated as the Klippel-Feil syndrome falls in this group. List has described three mechanisms to produce the neurological syndromes.

1) The bony deformities produce mechanical compression of the neuraxis,
2) There may be an associated congenital independent malformation of the nervous system,
3) A combination of the above two may occur.

The Bony deformities which occur are·

1) Malformation of the occipital foramen, basilar compression into posterior cerebral fossa, platybasia
2) Fusion of the atlas with occiput associated with dislocation of atlas on the axis.
3) Fusion of the other cervical vertebra - Klippel-Feil.
4) Abnormal position of axis in relation to occiput and atlas.

One case of this type will suffice to illustrate the diagnostic features.

A twenty-one-year-old man, two days after pitching baseball developed numbness of right leg and arm and two days later weakness and dizziness with dysphasia. A diagnosis of tuberculosis of the cervical spine was made and a laminectomy done. X-rays showed invagination of the base of the occiput with cranial displacement of the cervical spine He now had symptoms of ataxia in the arms and legs and a cerebellar gait, symptoms produced by intracranial irritation and tract compression by long pressure, true platybasia

(3) Pathologic lesions of the vertebra may produce both referred shoulder and arm pain or direct nerve-root irritation. It is also possible, of course, for the lesion to extend and produce cord pressure with tract syndromes.

Pyogenic infection such as acute purulent infection, osteomyelitis of the cervical spine may occur. Tuberculosis or other granuloma such as actinomycosis may produce destructive lesions of the cervical spine. The clinical signs of muscle spasm, limited motion of the cervical area plus the characteristic roentgen changes of inflammation, principally the evidence of bone destruction associated with intervertebral disc narrowing, aid in substantiating the diagnosis.

Arthritis of the cervical spine occurs so frequently that changes between the fifth and sixth cervical bodies, chiefly narrowing of the disc space and osteophytic formation on the contiguous vertebral edges, are regarded as possibly physiologic.

More extensive evidence of arthritis is seen, however, in a number of patients with osteophytic proliferation around the intervertebral foramina, narrowing of the several disc spaces and facet changes. These people frequently will be relieved of their symptoms by halter traction. Sometimes they require a cervical collar to immobilize the neck for a time.

The ankylosing type of arthritis produces symptomatology with radiation of pain during the acute phase of the lesion until ankylosis results. When the fixation of the spine is complete, the pain usually subsides. This fact seems to rule out bony pressure at the vertebral foramina as a cause of the referred pain.

Primary bone malignancy of any type can produce destruction in the vicinity of the nerve roots or produce symptomatology by referred pain. I refer to a case of chondrosarcoma of the cervical spine It is well always to bear in mind the possibility of malignancy, although it is relatively rare in this area. Myeloma is another potential cause for primary malignancy in the cervical area. The pain in these cases is quite different from that in the less serious type. The pain is intense, worse at night, and rarely relieved by position Secondary metastatic malignancy may occur in the cervical spine with severe symptoms of cervical pain radiating into the arm. We cite a case which demonstrated extensive destruction of the third cervical from a metastatic breast malignancy.

(4) Pancoast tumor. Superior sulcus tumor. The following is a case report. W. G. age 73. Male History of onset of pain three months ago with pain in his left shoulder, in the scapula and deltoid area radiating down the left arm to the elbow The pain also radiated on to the anterior chest wall. He applied immediately to competent medical men. X-rays of the shoulder were negative Physio-therapy did not relieve. The pain increased in severity, nocturnal in type.

He presented marked atrophy of the left shoulder girdle and spastic muscles Very little evidence of limitation of shoulder joint motion was observed. X-ray of the chest demonstrated a definite mass in the superior sulcus associated with a destructive lesion of the transverse processes of the upper dorsal vertebrae and the ribs at the costobertebral junction. The sedimentation rate was high, 1 1 , hematocrit 38. There was no definite evidence of a true Horner's syndrome in this case, which is the most frequent guide to Pancoast's tumor clinically.

(5) We wish here to pay particular tribute to Semmer and Murphy who in 1943 stressed the syndrome of lateral rupture of the cervical intervertebral disc. Since their original work, they have more recently reported thirty-two cases verified by operation with one hundred and six cases presenting x-ray and clinical findings characteristic of the lesion. The usual history of onset is akin to that recognized in intervertebral rupture producing sciatica

1) Recurrent attacks of stiff neck. Crick in the neck.
2) Later, months or years, pain located in the cervical spine, the shoulder, the anterior chest wall, the medial border of the scapula, and then radiating down the arm The pathway is segmental and dependent upon the nerve root involved.

The sixth and seventh cervical root nerves are the most commonly involved. Weakness of the grip, vasomotor changes, edema of the hand may occur. Sensory changes of the cutaneous type are in the segmental skin fields. Reflex changes may be present, C-5 supplies the biceps and C-6 the triceps. Point tenderness over the affected nerve root on percussion or compression is almost pathognomonic according to Semmer.

Spurling places great confidence in the compression test, tilting the head toward the affected side and putting pressure on the top of the head.

The x-ray shows evidence of

1) Reversal normal cervical concavity,
2) Narrowing disc space,
3) Calcification and spur formation, anterior and posterior,
4) Foramina narrowing,
5) Myelogram defects.

Operative removal of the extruded disc often produces brilliant results

Intervertebral foramina lesions are potential factors for the production of brachialgia Sicard called the intervertebral foramina the crossroads for sciatica. Oppenheim, Josters, Nachlas, and others have pointed out the strong probability of frequent involvement from arthritis. The irritation of the nerve roots might by direct pressure or by inflammatory reac-

tion, be within the intervertebral foramina.

Recently Arthur Davis pointed out the loss of normal cervical curves in patients with brachialgia Flexion films in his cases would frequently demonstrate subluxation of the cervical facets. I am sure, as time goes on, more attention will be paid to facet pathology as a cause for both sciatica and brachialgia.

Extension deformities of the cervical spine described by Lloyd Brown as the results of faulty posture can be eliminated by correcting posture. Brown states that often this will produce relief of the neurological and vascular changes which are seen in the so-called scalenus anticus syndrome.

Pressure in the supraclavicular region may be produced by

1) The Scalene syndrome, Naffziger, Oschner, Sage.
2) Narrowing of the costoclavicular space; Falconer and Waddell.
3) Cervical rib.
4) Aneurysm of the subclavian vessels.

The scalene syndrome is to our mind a real lesion, due to contracture and hypertrophy of the scalene anticus muscle which, however, is very probably secondary on many occasions to other irritative phenomenon, a secondary lesion. It is possible to have vascular and neurological changes with this syndrome. The cervical rib may be present or absent. The symptoms in one series of cases·

1) Pain of variable intensity.
2) Numbness Paresthesia. Tingling.
3) Tenderness supraclavicular area.
4) Slight to marked motor weakness. 14 of 21.
5) Atrophy. 14 of 21.
6) Reflex changes. 7 of 18.
7) Sensory changes. Lower portion of plexus only involved.
8) Vascular changes. Lowered blood pressure. Obliteration of pulse. Head toward side and deep inspiration.

Not infrequently there is a pseudo or aneurysmal dilation of the subclavian artery below the compression.

Operative resection of a portion of the scalenus anticus has been accompanied by relief. Gage has in fact demonstrated relief by novocaine injection into the scalene muscle.

Recently the pectoralis minor has been regarded as a factor in producing vascular cut-off when soldiers stand at attention or an abduction of the arm producing vascular symptoms, even a mild gangrene.

Lesions about the shoulder frequently produce radiation of pain proximal as well as distally in accordance with the root field and cutaneous field of the involved area. There are usually observed definite local findings in the shoulder, such as limitation of motion, loss of the normal rhythm of movement, associated with areas of tenderness and other clinical signs corroborated by x-ray evidence of calcification, or localized osteoporosis, or other suspicious evidence. The chief lesions to look for are periarthritis of the shoulder capsule due to bicipital tendinitis. Subdeltoid bursitis without evidence of calcification can occur. Calcification of the supraspinatus tendon or other structures about the shoulder are fairly common cause for brachialgia. Bosworth found that twenty per cent of several thousand people presented fluoroscopic evidence of this lesion. Rupture of the supraspinatus tendon may produce the classical picture described so well by Codman or may present an obscure brachialgia. Recognition of the true pathology can be determined, however, by careful observation of loss of normal function of the shoulder with usually a non-neurogenic brachialgia.

I can not leave this group of factors producing brachialgia without a warning of the frequency of a psychoneurotic personality developing a mild somatic cause for brachialgia with the psychic factors developing out of all reason to the minimal somatic factors. A careful recognition of the psychosomatic problem should confront us in every patient with predominant subjective findings.

In conclusion, a differential diagnosis may lead us to a definite diagnosis of the lesion which produces brachialgia. What can be done to cure it? With full recognition of the causative factors productive of the lesion, one has a most optimistic opportunity for a cure of the symptomatology, except for the neoplasms.

We have attempted in this paper to explain the mechanisms of radiation of pain, whether the lesion is neurogenic or non-neurogenic. Pain in itself is not diagnostic. Treatment must be directed toward the causative factor of the syndrome. There is no single cause for brachialgia but there are many individual lesions which can produce this syndrome.

REFERENCES

Inman, V. T , and Saunders, J B Referred Pain from Skeletal Structures. J. Nerv and Ment Dis , 99 660-667 (May, 1944)

Nachlas, I W. Brachialgia Manifestation of Various Lesions J. Bone & Joint Surg. 26 177-184 (Jan , 1944)

Moseley, H S. Shoulder Lesions Springfield, Ill., Charles C. Thomas, 1945.

Course No. 6

LOW BACK LESIONS

Lecturers

Halford Hallock, M.D., "Low Back Lesions - Anatomical Considerations"

Albert B. Ferguson, M.D., Roentgenography of the Lumbosacral Area

Halford Hallock, M.D., The Surgical Treatment of Low Back Pain

Paul C. Williams, M.D., The Diagnosis & Conservative Management of Lesions of the Lumbosacral Spine

LOW BACK LESIONS - ANATOMICAL CONSIDERATIONS

Halford Hallock, M.D.

IN ORDER TO UNDERSTAND clearly the basis for mechanical low back pain with or without sciatica, it is essential to appreciate certain anatomical features and variations.

The lumbosacral articulation is placed at the meeting of two natural curves of the spinal column and at the junction of a mobile portion with a relatively immobile one. Through this joint to the pelvis is transmitted all the weight of the arms, head, and trunk. Great strain, therefore, is localized in this area. In many individuals this strain is exaggerated by the presence of developmental or degenerative variations of structure which impair the mechanics of this joint.

The Lumbosacral Angle

In the upright position the sacrum is inclined forward so that its superior surface, upon which the 5th lumbar vertebra is supported, makes an average angle of 42^{o} with the horizontal. Considerable variation in this angle occurs, and when it is increased, the shearing strain on the ligaments and intervertebral disc is made proportionately greater. Also, with exaggeration of the lumbosacral angle there is generally an increase in the lumbar lordosis. The center of gravity which usually passes vertically through the body of the 3rd lumbar vertebra then lies at a considerable distance anterior to the sacrum, further increasing the strain.

The lumbosacral angle, provided that no contractures exist in the lumbar spine or anterior portions of the hip joints, can be altered by action of the muscles that control rotation of the pelvis and sacrum. These are the sacrospinalis and the hip flexors, which rotate the pelvis backward and increase the angle, and the gluteus maximus and the abdominal muscles, which rotate it forward and reduce the angulation. Development in the use of the latter muscles is important,. therefore, in the therapy of low back pain arising from postural strain.

The Posterior Articulations

Normally these are concavoconvex and allow a limited amount of gliding motion centered in the intervertebral disc. They usually lie in an oblique plane or halfway between the sagittal (internal-external) and coronal (anteroposterior) planes. Variations, however, are frequent. They may be underdeveloped, defective, or rudimentary, and may lie in any one of various planes. Those that are internal-external in type or in the sagittal plane are considered to be the best mechanically as they allow flexion and extension but check lateral flexion and rotation. Those that have developed

in the oblique or in the coronal or transverse plane are not so strong, for they allow a larger amount of motion in all directions including rotation, thereby creating unstable conditions. Asymmetrically developed facets or those in dissimilar planes give rise to extremely faulty mechanics because they operate in different planes. Consequently one or the other is constantly being strained by motions of the trunk.

Sacralization

Possibly because of an evolutionary attempt to increase stability for the upright posture by shortening the lumbar spine, the 5th lumbar at times is incorporated in the sacrum. This is of importance in relation to low back pain when this incorporation or sacralization is incomplete on one or both sides. When this is the case, the enlarged transverse processes of the 5th lumbar make contact through a false joint with the lateral masses of the sacrum. A narrow intervertebral disc is present and a limited amount of motion occurs through arch articulations that are small and rudimentary. Strains are likely to occur under these adverse mechanical conditions.

Posterior Subluxation of the 5th Lumbar Vertebra

Posterior subluxation arises from a hypermobility or instability and therefore is usually seen in association with anteroposterior, oblique, or asymmetrical articulations which favor increased motion In the backward movement of the vertebra the inferior articular processes impinge against the sacrum and at operation sacral hollows have often been found into which these facets have subluxated. Also in the posterior movement, the vertebral body encroaches upon the intervertebral foramina and the enclosed 5th lumbar nerve roots, which is very likely the explanation of the frequent sciatic pain that is encountered in this condition.

Spondylolisthesis

This is a forward displacement of the 5th lumbar vertebra resulting from developmental defects in the interarticular portions of the vertebral arch. It has been demonstrated that the arch is formed by the union of two ossification centers for either side, one for the pedicle and superior articular process and one for the lamina and inferior process. Failure of these centers to unite leaves the body of the vertebra unattached by bone to its neural arch. Marked instability of the lumbosacral joint is the result. While examples of this defect have been seen in other lumbar vertebrae, it is most frequent in the 5th.

The Intervertebral Disc and Nucleus Pulposus

Between each vertebral body there is interposed a fibro-cartilaginous disc in the center of which is enclosed a semigelatinous incompressible substance which is the nucleus pulposus. Motion between the vertebrae is centered in the nucleus and is controlled and guided by the gliding posterior articulations. The nucleus also acts as a hydrostatic shock absorber in transmitting pressure stresses in the spinal column. Loss of integrity of the nucleus or disc interferes with this normal pattern of motion and buffer action and produces serious mechanical effects with impairment of function.

The discs in the lower lumbar region, particularly the lumbosacral one, are under greater stress and strain than any of the others If in addition any anatomical abnormality is present, the strains may be even greater. Therefore it is not surprising that frequent traumatic or degenerative changes are seen in these structures in this area.

Loss of normal function may occur in two ways. The disc may become narrowed following atrophy or degeneration of its substance or the nucleus may be lost as a result of protrusion into the cancellous vertebral bone through a defect in the cartilage plate (Schmorl's nodules) or herniation through a rupture of the enclosing annulus fibrosus. In the latter case, posterior herniation is more important because of nerve root pressure and consequent sciatica.

The Intervertebral Foramina and Nerve Roots

The intervertebral foramina transmit the spinal nerve roots as they pass outward from the intraspinal canal. As the nerves emerge, the posterior primary divisions turn backward into the spinal muscles and innervate the articulations. *Each foramen anteriorly* is formed by the intervertebral disc and the upper and lower borders of the adjoining vertebrae, superiorly and inferiorly by the pedicles, and *posteriorly* by the articular processes. The nerve roots, therefore, are in position easily

to be influenced by any condition involving the spinal muscles, articulations, the intervertebral disc, or the relative positions of the vertebral bodies to each other. Although the lumbosacral foramina are the smallest of the lumbar series, they transmit the largest of the lumbar nerve roots. It is believed that these anatomical relationships may explain the occurrence of sciatica in the low back syndrome when it is not produced by a posterior nuclear herniation.

The Sacro-Iliac Joints

These articulations, which lie in the low back area, are exceptionally firmly constructed and are well supported by ligaments. The degree of mobility is small. Also, they are not subject, as is the lumbosacral region, to anatomical variations which modify mechanics. For these reasons it is believed that symptoms arising from mechanical strains or instability of these joints must occur but rarely. This is borne out by clinical experience.

From a consideration of the above anatomical features and structural variations, the mechanical basis for low back pain and sciatica is understood more clearly. The roentgenography will be discussed in the next paper.

REFERENCES

1. Danforth, M. S., and Wilson, P. D. The Anatomy of the Lumbo-sacral Region in Relation to Sciatic Pain. J of Bone & Joint Surg. 7 109-160 (1925).
2 Ferguson, Albert B. The Clinical and Roentgenographic Interpretation of Lumbosacral Anomalies, Radiology 22.548-558 (1934).
3. George, Everett M. Spondylolisthesis. Surg. Gynec & Obst 68 774 (1939)
4 Hibbs, Russell A , and Swift, Walker E. Developmental Abnormalities at the Lumbosacral Juncture Causing Pain and Disability. Surg., Gynec. & Obst. 48 604 (1929).
5 Lackum, H. L. von The Lumbosacral Region, J A. M. A. 82 1109 (1924).
6 Smith, Alan De Forest The Surgical Treatment of Low Back Pain, Surgery 4 13-20 (1938)
7. Willis, T A. The Separate Neural Arch. J. Bone & Joint Surg. 13 709 (1931).

ROENTGENOGRAPHY OF THE LUMBOSACRAL AREA

Albert B. Ferguson, M.D.

Dr. Hallock has described and illustrated the roentgen appearance of anomalies associated with low back pain. The roentgen examination and interpretation of these anomalies is now the subject of discussion.

Posterior protrusions of an intervertebral disk into the neural canal are not directly visible in the roentgenogram but are indirectly visualized by injection of contrast material. The technique is too well known to require detailed description here. It suffices to say that injection of lipiodol results in the most definite and readily interpreted contrasts, that fluoroscopic examination is essential with tilting and turning of the patient to flow the opaque material into various portions of the neural canal, that the positions most likely to yield positive findings are the prone and prone oblique positions, that protrusion of a disk is indicated by the presence of a rounded area at or just above the level of an intervertebral disk into which the opaque material cannot be made to enter; that the defect seen fluoroscopically can be recorded on films with the position of the patient unaltered, and that lipiodol should be removed by aspiration when the examination is concluded.

Roentgen examination for lumbosacral anomalies is essentially an attempt to demonstrate abnormally close relation of bony parts (which may cause pressure), displacements, and the elements of lumbosacral instability, which are unstable lumbosacral angle, unstable facets, and various degrees of spina bifida.

These objectives are attained only when the relationship of the bones is truly shown. For

this reason it is essential that the routine examination should be based upon a true lateral and a true anterior-posterior view of the lumbosacral area. These views are best obtained *in the lying position* not only because it is technically easier but also for other reasons which will appear in the discussion of the lumbosacral angle. An anteroposterior view oblique from below upward in the plane of the lumbosacral joint (about forty degrees of tilt) is so generally useful for the demonstration of the relation of the transverse process of the fifth lumbar vertebra to the sacral wings and ilium, for the sacroiliac joint and for defects in the arch of the fifth lumbar vertebra that it should be included in the routine examination of the lumbosacral area.

Other views should be added to the routine examination when specific conditions are suspected or must be eliminated. In this category are oblique views in the plane of the arch articulations or the sacroiliac joints, views in the erect posture (to be discussed in connection with the lumbosacral angle), and views with the patient bent to the right or left side or in flexion or extension.

The views with the patient bent passively *to the limit of motion* are used chiefly to determine the solidity of fusion or of ankylosis but they may also be used for another purpose. It is unlikely that a patient having posterior protrusion of an intervertebral disk will bend equally to the right and left at the joint affected. These views may therefore be used to determine the probable absence of protrusion of a disk.

Close relation between spinous processes, between a spinous process and a lamina, between the transverse process of the fifth lumbar vertebra and the sacral wing or ilium as seen in the anteroposterior view oblique from below upward, or between bony parts about the neural foramina are clearly seen and readily interpreted in true examinations. Displacements, unstable facets, and unstable lumbosacral angle require some discussion.

Displacement of the fifth lumbar vertebra anteriorly or posteriorly relative to the first sacral vertebra is not easily measured. The roentgenogram must be a true lateral view. In such a view the best base line for measurement of the displacement is a line joining the posterior superior angle of the body of the fifth lumbar vertebra and the posterior inferior angle of the body of the first sacral vertebra. If a line parallel to this base line be drawn at the posterior superior angle of the body of the first sacral vertebra the deviation therefrom of the body of the fifth lumbar vertebra represents the amount of displacement. The method loses accuracy when displacement is very great, but then the need for accuracy has vanished.

Arch articulations may be unstable when they are underdeveloped, poorly formed, tilted laterally, asymmetrical, or of a type other than the *internal-external*. Judgment of the type facets is readily made on the anterior-posterior view. In the internal-external or stable type the individual facets of each pair face each other from side to side, the area of overlap *of the shadow* of the fifth lumbar and the first sacral facet is not more than *one* quarter of the total width of the facet shadows. In the anterior-posterior or unstable type the facets face each other from front to back, the area of overlap of the *facet shadows is the full width* of the shadows. Various degrees of obliquity are represented by various amounts of overlap between these two extremes. Fifty per cent overlap represents about forty degrees of obliquity. *The instability most certain to result* in strain and pain at an early age is severe asymmetry of the facets with one side anterior-posterior, the other internal-external type.

The lumbosacral angle has two elements of instability each of which is readily judged in the true lateral view. This view should be obtained in the lying position in the habitual posture of the patient with the hips normally extended. The patient lies in the long axis of the table, which is the same as the axis of the film. The bottom of the film therefore represents the horizontal for the patient.

The lumbosacral angle is unstable if the inclination of the superior surface of the first sacral vertebra is more than forty-two degrees to the horizontal as represented by the bottom of the film. Fifty-two degrees of inclination represents severe instability.

The lumbosacral angle is also unstable if the center of gravity for the trunk, as represented by a vertical line (perpendicular to the bottom of the film), through the center of the shadow of the body of the third lumbar vertebra passes more than one-half inch anterior to the anterior superior angle of the sacrum. If this line is more than one and a half inches

anterior to the sacrum instability is severe. Both of these elements of instability may be present at the same time.

The reason why the examination for stability of the lumbosacral angle should be made in the lying position is this On standing, the pelvis normally tilts forward, increasing the lumbosacral angle. When lumbosacral strain is present the opposite reaction occurs, the pelvis tilts backward bringing the sacrum more nearly under the line of weight thrust and reducing the lumbosacral angle. Therefore, on standing, the unstable lumbosacral angle may appear more stable than it actually is and may appear stable. The examination lying with the patient in habitual posture yields the true picture.

The detection of lumbosacral anomaly or instability in the roentgenogram ordinarily indicates merely the presence of a mechanical weakness which may predispose to lumbosacral strain. In some cases it is possible to prove the actual presence of such strain at the time of examination. If the lateral views lying and standing be compared and it is found that the pelvis tilts backward and the lumbosacral angle is reduced on standing, this protective reaction is definite evidence of the actual presence of lumbosacral strain. This may be a very useful examination when malingering is suspected.

The various degrees of spina bifida contribute to other weaknesses at the lumbosacral junction by necessitating elongation of the ligaments but are not by themselves a potent source of lumbosacral strain. One form of this anomaly is peculiar. The absent spinous process may be represented by a rudimentary ossicle. In some cases this ossicle may be united to the spinous process of the fifth lumbar vertebra, prolonging it downward between the sacral laminae. Pressure caused by this long spinous process on motion of the fifth lumbar vertebra may cause much discomfort and be relieved only by excision of the elongated process.

The defect which permits forward slipping of the body of the fifth lumbar vertebra in spondylolisthesis is failure of union of the laminae to the pedicles. This defect is always present or has been present (it may become reunited after thirty-five years of age). The defect may possibly be visible in any view of the lumbosacral area but the view which is most dependable for its demonstration is the anteroposterior view oblique from below upward (commonly called the forty-five degree view).

This defect may be claimed to be a fracture in some legal cases. Some of the other features of the condition are rudimentary articular facets, underdeveloped arch of the fifth lumbar vertebra, and broadening of the anteroposterior diameter of the body of the first sacral vertebra. Each of these changes is a variation of development which occurred only during the years of growth in height of the spine. The presence of any one of these changes establishes that the condition dates back to childhood.

The defect which is responsible for spondylolisthesis is not found in the fetus or newborn with sufficient frequency. It is not a congenital defect and yet it is developmental. An unproved but plausible theory to fit this situation is the following The neural canal enlarges during growth. Enlargement of the vertebral arch about the neural canal is accomplished by resorption of bone internally and accretion externally. The accretion is a response to stress. The tilted position of the fifth lumbar vertebra at the lumbosacral junction reduces stress on its arch and reduces accretion. Resorption tends to catch up with accretion. When it does so the lamina is no longer united to the pedicle and spondylolysis has developed.

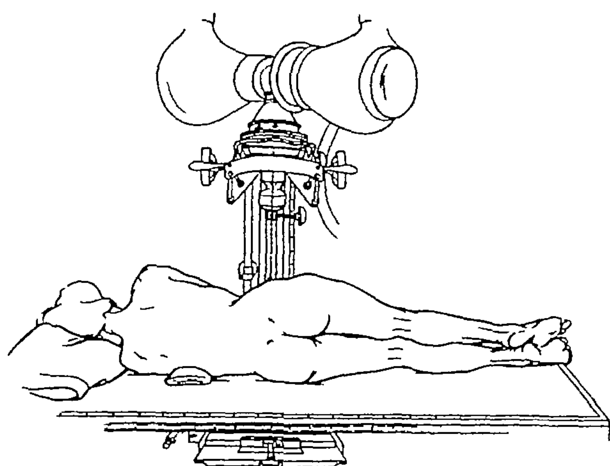


Fig 1 Position for lateral view, lying.

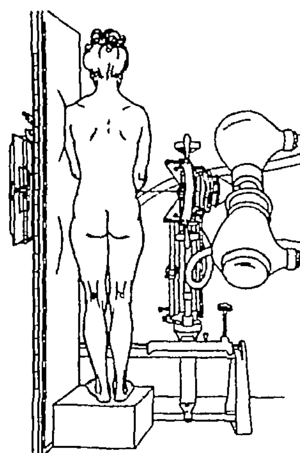


Fig 2 Position for lateral view, standing

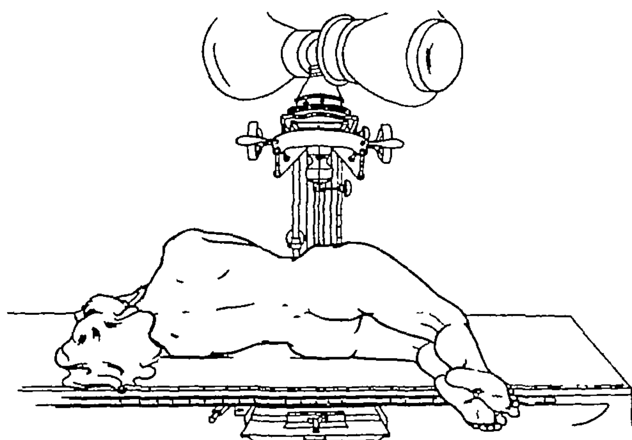


Fig 3 Position for lateral view, extended

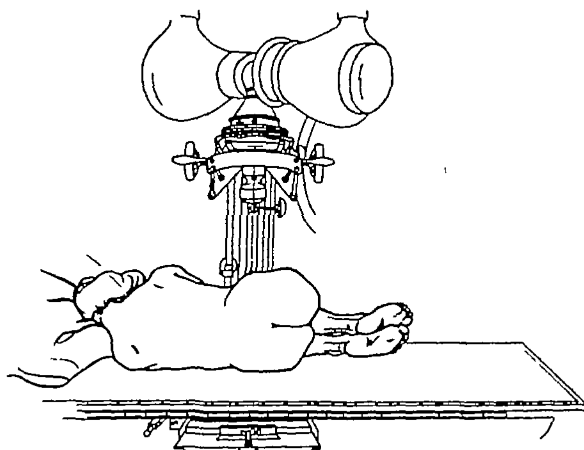


Fig 4. Position for lateral view, flexed

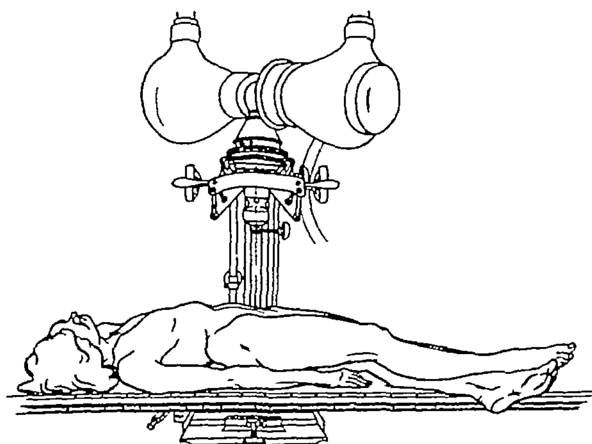


Fig 5. Position for bending to the right

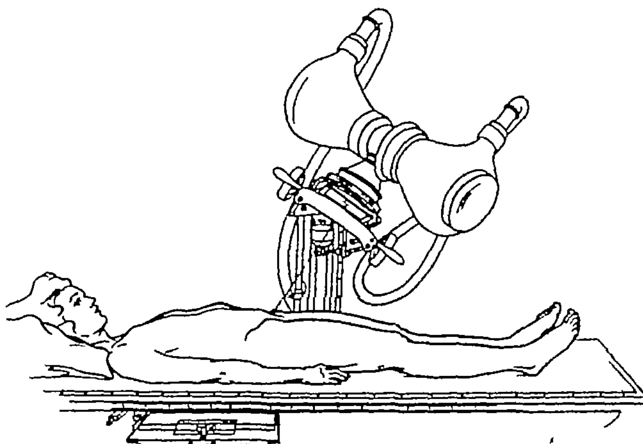


Fig 6 The anteroposterior view oblique from below upward. See also Fig 8.

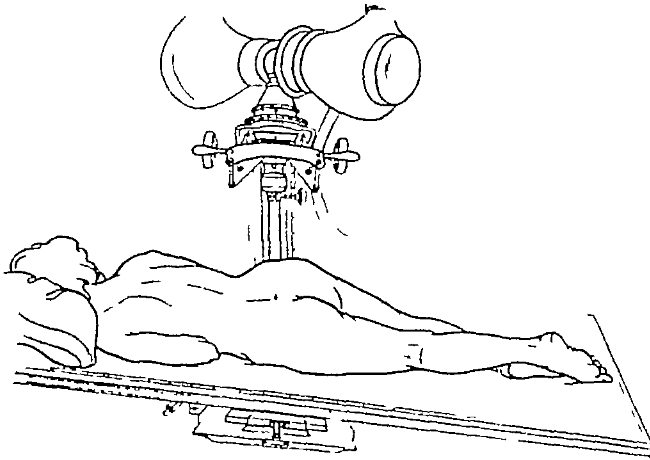


Fig 7 The oblique view on the tilted table for spinagram.



Fig 8 The anteroposterior view oblique from below upward showing failure of union of the laminae to the pedicles (at arrows), the close relation of the lumbar transverse processes to the sacral wings, and the normal state of the sacral wings. See also Fig. 6.



Fig 9. Defect of lipiodol shadow at posterior protrusion of the intervertebral disc at the lumbosacral joint

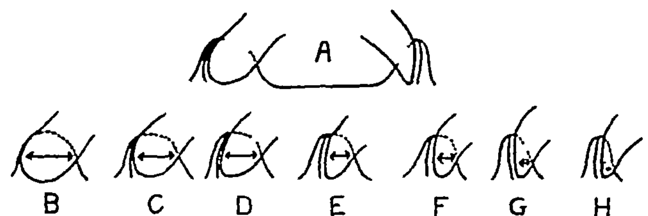


Fig 10 Diagram of facet shadows at asymmetrical arch articulations (A) and of overlapping of facet shadows ranging from complete overlap at anteroposterior type facets (B) to little overlap at internal-external type facets (H)



Fig 11 The lumbosacral facets The long line indicates the total width of the facet shadows, the short line the amount of overlap of the facets The plane of the joint is oblique



Fig 12 The base line drawn posteriorly from the proximal angle of the fifth lumbar to the distal angle of the first sacral vertebral bodies and the short lines parallel to the base line which demonstrate the normal alignment of those vertebrae

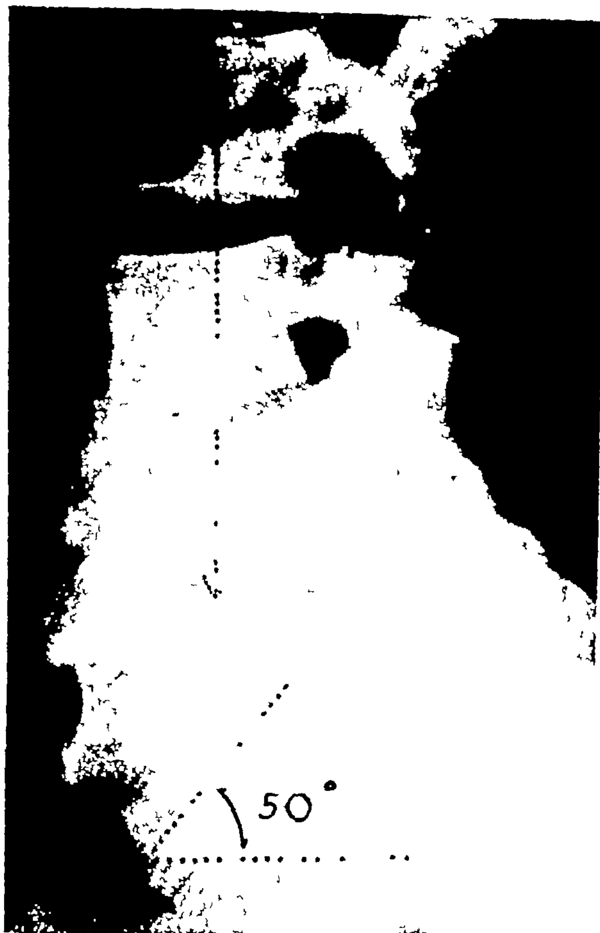


Fig 13 The lumbosacral angle, 50 degree inclination of the proximal sacral surface to the horizontal Also, the line of weight thrust, the vertical line through the center of the third lumbar vertebral body passes about one inch anterior to the sacrum.

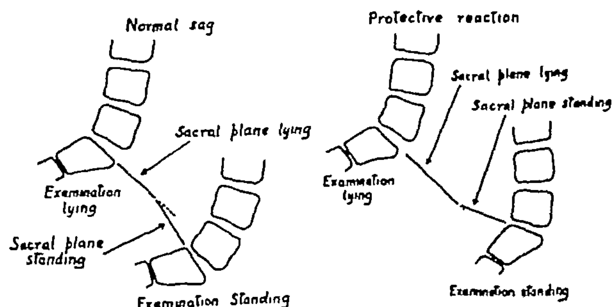


Fig 14. Diagram of normal sag and of protective reaction as seen in lateral views lying and standing.

THE SURGICAL TREATMENT OF LOW BACK PAIN

Halford Hallock, M.D.

Although the great majority of patients suffering from low back pain of mechanical origin are treated successfully by nonoperative means, there is a small number, about 10%, that continue to have more or less severe chronic or recurrent symptoms and varying degrees of disability.

Uncompensated muscular and ligamentous strain is the cause of the pain, and it is believed that this is related to the developmental or degenerative structural variations that often are found at the lumbosacral joint. These variations consist of abnormality of the lumbosacral angle, alterations in the shape, size and plane of the posterior articulations, transitional vertebra or sacralization, posterior subluxation of the 5th lumbar vertebra, atrophy of the intervertebral disc, and spondylolisthesis. By creating mechanical abnormalities of relationship and support, these variations produce abnormal stresses and strains. When these can no longer be fully met or compensated for by the muscles and ligaments, operative treatment with spine fusion is indicated.

Lumbosacral Spine Fusion

Lumbosacral spine fusion is a rational procedure, for it eliminates mobility and creates stability in a joint that is mechanically inadequate and strained. It is indicated when nonsurgical methods have failed or when the structural changes disclosed by the x-ray are so great as to preclude the possibility of otherwise achieving a satisfactory and lasting result. Before advising operation, it must be reasonably certain that the symptoms arise from a mechanical cause and not from some condition not amenable to spine fusion such as an early Marie-Strumpell spinal arthritis or a neoplasm. In addition, the patient should be in adequate physical condition and in good mental health, and preferably not involved in a long and complicated legal or compensation suit. With these precautions and when performed by experienced surgeons, the operation yields a high percentage of good and excellent results.

At the New York Orthopedic Hospital, the Hibbs type of spine fusion is used exclusively. Several minor modifications have been added to the original technique but the basic principles are the same. The present technique has been well described and illustrated by Howorth in 1943[1] and need not be repeated here. Since 1944, however, internal fixation has been employed almost routinely in lumbosacral cases in addition to the regular fusion technique. This is accomplished by passing one and one-quarter inch stainless steel or vitallium screws through both lateral articulations down into the substance of the sacrum in a direction of 30 to 35° downward and lateralward. Fixation of the 4th and even of the 3rd lumbar similarly has been secured by one-inch screws which pass downward and outward into the area of the transverse processes of the lower vertebra.

With such internal fixation it has been possible to dispense with postoperative support and to allow the patients up much earlier, usually 10 to 14 days after operation. In spondylolisthesis, however, a brace still is used and recumbency for six weeks is required because the screws do not provide fixation of the neural arch defect and some motion still exists between the posterior elements of the 4th and 5th lumbar vertebrae.

Several points in the operative technique should be emphasized. The exposure of the spinous processes, laminae, and articulations must be subperiosteal in order to minimize hemorrhage. Should troublesome bleeding occur, clamp or pack off the injured vessels and work in another part of the field until the bleeding has been controlled. The ligaments and periosteum should be removed completely from the spinous processes and posterior surfaces and edges of the laminae.

Removal of the cartilage from the articular facets should be as complete as possible. Small angled osteotomes and curets are used for this. The cartilage surfaces are larger than is usually thought and painstaking care is required to denude them completely. It is well also to remove the thin underlying layer of cortical bone as fusion probably will more rapidly and surely take place between two can-

cellous surfaces. The resulting spaces, which should not be too wide, are filled with thin bone grafts which, when internal fixation is used, will be held in place by the metal screws as they pass across the articular areas.

In the actual fusion an adequate number of chips, or better, bony bridges, which are left attached at one end, should be turned up and down from the posterior arches in an interlacing fashion to overlie and make contact with the upper and lower laminae. The articular fossae and processes should be well filled and covered with chips. If the supply of bone from the spine is insufficient for this purpose, additional bone should be removed from the posterior iliac crest or tibia. In raising the bony bridges or removing chips from the laminae, too much bone should not be removed for otherwise the viability and integrity of the remaining portion of the arch may be compromised and the development of fusion jeopardized.

Methods of fusion employing massive bone grafts, such as the Albee, have been described and unquestionably are successful in a large number of cases. Massive grafts, however, often undergo absorptive changes, and because of their size require a long time for replacement by new and living bone The lumbosacral curve and the usually small size of the 1st sacral spinous process increase the technical difficulties of securing adequate and close application of such a graft. Bosworth employs a "clothespin" graft which is locked between the 5th lumbar and 1st sacral spinous processes under distraction so that it provides fixation and at the same time increases the vertical diameter of the intervertebral foramen Small bone transplants, which is the essence of the Hibbs method, are much more quickly rehabilitated than massive bone grafts and are more effective as agents of osteogenesis It is believed that they should always be used in a spinal fusion even though for some reason or another it is deemed advisable also to employ a massive graft Fusion of the articulations themselves is an integral part of the Hibbs procedure, and for this reason, too, this method is considered to be superior

Post-Operative Treatment

Unless internal fixation is used, a Taylor spine brace is applied on the operating table and is worn continuously for from 3 to 4 months If x-rays at six weeks show a satisfactorily developing fusion, the patient is allowed up. Generally he may return to light or sedentary work in 3 to 4 months and to heavy work in 6 months. If screw fixation is employed, a brace is not used and the patient may be allowed up 10 to 14 days after operation. Often he will be able to return to work, except heavy labor, in 8 to 10 weeks. In very large and fleshy individuals, when external support is required, a plaster jacket-spica including both thighs is employed This is thought to give better immobilization than a brace under these circumstances.

Complications

Mortality

1,403 patients with low back pain with or without sciatica have had lumbosacral spine fusions at the New York Orthopedic Hospital from 1914 through 1946. Three of these, or 0.002%, died from causes directly related to the operation. Two deaths occurred from streptococcus hemolyticus septicemia secondary to wound infection and one from a postoperative pneumonia. There have been no deaths in this category since 1935.

Pseudarthrosis

Failure of fusion or pseudarthrosis is the most frequent cause of failure to relieve the patient's back pain in properly selected and correctly diagnosed cases. While we have seen a few instances of failure of bone to grow, it is believed that the development of pseudarthroses is due in large part to faulty operative technique or inadequate postoperative immobilization

The percentage failure of fusion at the New York Orthopedic Hospital from 1931 through 1938[2] was as follows

Fusion L5 to Sacrum - 276 patients - 7 9% pseudarthrosis
Fusion L4 to Sacrum - 72 patients - 23.6% pseudarthrosis
Spondylolisthesis - 62 patients - 14 3% pseudarthrosis

The possible presence of a pseudarthrosis always must be considered in a patient who continues to have back pain after a fusion op-

eration. Sciatica sometimes may be present as well. Frequently routine anteroposterior and lateral x-rays will fail to disclose the defect because of overlapping bone shadows in this region or on account of obliquity of direction and plane of the area of nonunion. Oblique views or laminographs may be helpful. Of greatest aid in cases of doubtful interpretation are true lateral x-rays taken in as nearly comparable positions as possible, first with the hips and trunk extended, and then flexed. If a pseudarthrosis is present, usually a little motion can thus be demonstrated in the fusion area Recently, however, because of continuing pain, a fusion was explored although the flexion and extension views showed no evidence of movement. A pseudarthrosis with very little motion was found. Either this was too small in amount to show in the x-rays or the patient at the time of examination had so much pain and muscle soreness that she could not flex and extend sufficiently to produce an appreciable amount of motion through the defect.

Repair of fusion failures should be ,undertaken unless symptoms are minor or absent. In general the fibrous tissue is removed from the defect, the bone edges and surfaces are freshened and the area is filled in and covered with numerous small bone transplants taken from the ilium or tibia. A successful result is obtained in close to 95%.

Sciatica

This is a frequent complication of low back pain. In earlier years it was thought to be due to mechanical or congestive irritation of the nerve roots secondary to mechanical low back strain The nerve roots are in close relation to the spinal joint which form the posterior walls of the intervertebral foramina. The posterior primary divisions after exit of the roots turn backward into the sacrospinalis muscles and innervate the posterior articulations. The lumbosacral foramen is the smallest of the lumbar series but transmits the largest of the lumbar roots. The nerves, especially the fifth, are therefore in a position where they could easily be influenced by congestive changes in the muscles and joint resulting from strain or by alterations in the mechanical relationship between the vertebrae affecting the intervertebral foramina.

In later years, it has been demonstrated by many observers that sciatica is frequently caused by a posterior herniation of the nucleus pulposus which presses upon the nerve root. The fact that many of these patients have had backache for months or years before the onset of their sciatica supports the contention that instability of the lower lumbar joints is a predisposing cause of the disc rupture.

Pressure upon the nerve root also can be produced by hypertrophy of the ligamentum flavum and a few instances of this have been seen. In several patients that were operated upon intraspinal scarring and arachnoid adhesions were found. Since there was no history of infection it was thought that these changes might have been related to a long-standing back strain.

Ober has demonstrated that in some, as yet unexplained manner, a contracted fascia lata may produce a sciatica. In these instances fasciotomy often affords relief. In a series of cases at the New York Orthopedic Hospital reported in 1937,[3] fasciotomy gave excellent or good results in 75% as far as the sciatic pain was concerned. Back pain was not similarly relieved. Before performing fasciotomy, one should be sure that no intraspinal lesion is present that might cause the sciatica.

Loss of the nucleus pulposus and consequent collapse of the disc has a serious effect upon the mechanics of the joint. This may account for the persistence of backache in many cases of removal of the herniation with relief of the sciatica. Experimental work has shown that interference with the normal function of the joint by destruction of the nucleus may be followed by osteoarthritic changes in the involved articulation. For these reasons and because lumbosacral instability appears to be a predisposing cause of disc injury, a fusion of the joint generally is performed at the time of removal of the herniation. In elderly individuals or those in poor health in whom the risk associated with a long procedure is too great, removal of the nucleus alone is proper.

At the New York Orthopedic Hospital, patients with low back pain and sciatica are examined both by an orthopedist and a neurologist. On the basis of the neurological examination they are divided into three groups - those in which the neurological signs are characteristic and convincing, those in whom the nerve signs are less definite and unconvincing and those in whom there is no definite evidence of an intraspinal lesion. In the first group, a def-

inite diagnosis of a herniated nucleus is made, in the second it is suspected and exploration of the nerve roots is advised especially if fusion has been decided upon for control of the low back pain. At exploration the 5L and 1S nerve roots should be explored in the 4th-5th L. and 5th L. to the 1st sacral interspaces, respectively, since a number of double discs have been found. A herniation in the 3rd-4th L space is believed to be relatively rare

Routine lumbar punctures with total protein determinations have not been found to be of great diagnostic value. These are valuable, however, in excluding suspected cases of spinal cord tumors.

Myelography with an absorbable opaque medium such as Pantopaque, is of value in certain cases where the diagnosis is greatly in doubt or the level of the lesion is otherwise indeterminable. As much as possible of the opaque material should be removed at the close of the examination Lipiodol is not favored as it is nonabsorbable and some instances of chronic arachnoiditis have been seen following its use. Air myelograms have not been satisfactory in our experience. Routine myelography is not considered to be necessary or advisable There have been a few instances in which such examinations have given negative results and yet at operation ruptured nuclei were found. Chief reliance is placed upon the history of sciatica and a careful neurological examination followed by nerve-root exploration in suspected cases having chronic or recurrent pain and disability.

The exploration of the nerve roots is performed after the laminae have been subperiosteally exposed. 1) The spinous processes are excised and cut up into chips to be used later on in the fusion. The ligamentum subflavum is removed, including the most lateral portion at the posterior boundary of the foramen. If necessary a little bone is removed from the edges of the laminae to obtain more room. The articulations should not be disturbed if a fusion is not contemplated. The unopened dura and the nerve root are carefully retracted toward the midline and the posterior aspect of the intervertebral disc exposed. Usually there are many veins in this region which tear easily. They are packed off with strips of moist cellucotton. The posterior longitudinal ligament over the bulging herniation is incised by a crucial incision and the protruding nuclear material is removed with the aid of pituitary forceps. A bit of crushed muscle placed over the injured veins and removal of the nerve-root retractor results in quick cessation of the bleeding. The fusion is then performed in the usual Hibbs manner, using internal fixation with screws and additional bone from the ilium when this is required. At the New York Orthopedic Hospital the orthpedist and the neurosurgeon generally work together as a combined team, the latter performing the intraspinal surgery and the former the fusion.

Postoperative care is the same as for uncomplicated fusion cases.

Exploration and removal of the nuclear herniation through the interspace in this manner sacrifices very little bone, and little difficulty is added to the fusion. The number of pseudarthrosis in these patients has been only 12%.

Infection of the Intervertebral Disc

In five or six of the patients, who were operated upon for the removal of a herniated nucleus, a low grade infection occurred postoperatively in the intervertebral disc. For several weeks after operation the course was not unusual but then pain increased for which no cause could be found. The temperature was not elevated and the wounds healed well. Usually there was no increase in the white blood cell count. Psychoneurotic factors were considered. The sedimentation rates, however, were uniformly increased. Early x-rays gave no clue, but in 6 to 8 weeks after operation, they demonstrated unmistakable evidence of an infectious process in the disc with bone destruction. Three patients were subjected to exploration and drainage. Diseased granulation tissue and pus were found in two, and in the third purulent material drained after a few days Cultures were negative in the first case, but in the second and third a nonhemolytic streptococcus and staphylococcus aureus respectively were grown.

All have recovered or are recovering satisfactorily, and those that have gone the longest have obtained more or less spontaneous bony ankylosis of the involved vertebrae.

Because of the possibility of this complication, it is considered inadvisable in operating upon the intervertebral disc to do more than remove the herniated nucleus and gently explore the disc with a curet to make sure *that*

all the nuclear material has been obtained. Extensive curettage in an attempt to produce fusion between the vertebral bodies would create a large cavity with rigid walls which in this relatively avascular region would predispose to the development of infection if any organisms were introduced. If bone chips also were inserted in the defect to promote a process of fusion, the situation would be made much worse in the event of infection by the presence of these extraneous "sequestra." Fusion readily can be obtained through the posterior elements without running any additional risk.

Results

Lumbosacral fusion has yielded a high percentage of excellent and good results. Reports from the New York Orthopedic Hospital have been published by Hibbs and Swift[4], Kimberly[5], George[6], and Smith, Deery and Hagman.[7]

In 1929, Hibbs and Swift reported 147 patients with an average follow-up of 4 years. Three had a second operation because of pseudarthrosis and so the report covers 150 operations. Seventy-three percent were entirely relieved, 14% improved, and 13% failed to obtain relief.

Kimberly in 1937 reported a group of 195 patients that had been followed for an average period of 5 years and 11 months. Some of these possibly had been included in the first report by Hibbs and Swift. Of these 78% obtained excellent or good results (excellent - 70.8%, good or 75 to 90% relief - 7.2%), 8.7% were relieved 50 to 75%, and 13.3% obtained little or no relief.

In 1939, George studied the results in 91 patients who were operated upon for spondylolisthesis with an average follow-up of 5 years. Complete relief of symptoms were obtained in 81.3%. Improvement occurred in 14.3% and unimprovement in 4.4%.

Smith, Deery, and Hagman, in 1944, reported 70 patients with sciatica who had been operated upon for removal of a herniated nucleus pulposus and spine fusion from January, 1937, to April, 1943, and who had been followed for at least one year after operation. With only one exception, all had had low back pain which in practically every case preceded the sciatica. Excellent or good results were obtained in 81% (excellent 34%, good 47%) and poor in 19%.

The major factors which reduce the percentage of good results are faulty diagnosis, pseudarthrosis, spinal arthritis, and compensation claims.

Sacro-Iliac Arthrodesis

The sacro-iliac joints are exceptionally firmly constructed and are well supported by ligaments. The degree of mobility is small. Also they are not subject, as is the lumbosacral articulation, to anatomical variations which modify mechanics. For these reasons it is believed that symptoms arising from mechanical strains or instability of these joints must occur but rarely. This is borne out by clinical experience.

Arthritis or even disease, however, may occur in these articulations and produce pain in the low back region for which sacro-iliac arthrodesis may be indicated.

REFERENCES

1. Howorth, M. B. Evolution of the Spinal Fusion. Ann. Surg. 117 278 (Feb., 1943).
2. Thompson, Walter A. L. Occurrence of Pseudarthroses in the Hibbs Spinal Fusion Operation - Read before the Orthopedic Section of the New York Academy of Medicine April 18, 1941 (not published)
3. Smith, Alan DeForest Results of Fasciotomy for the Relief of Sciatic Pain. J. Bone & Joint Surg. 19 765 (July, 1937)
4. Hibbs, Russell A., and Swift, Walker W. Developmental Abnormalities at the Lumbosacral Juncture Causing Pain and Disability Surg., Gynec. & Obst. 48 604-612 (May, 1929).
5. Kimberley, A. Gurnes Low Back Pain and Sciatica. Surg., Gynec. & Obst. 65 195 (Aug , 1937).
6. George, Everett M. Spondylolisthesis. Surg., Gynec. & Obst. 68 774 (April, 1939).
7. Smith, Alan DeForest, Deery, Edwin M., and Hagman, George L. Herniation of the Nucleus Pulposus - A Study of 100 Cases Treated by Operation. J Bone & Joint Surg. 26 821-828 (Oct., 1944).

Deery, Edwin M. Herniation of The Nucleus Pulposus as a Complication of Pre-Existing Low Back Instability Surg., Gynec. & Obst. 77 79 (July, 1943).

Ferguson, Albert B. The Clinical and Roentgenographic Interpretation of Lumbosacral Anomalies Radiology. 22 548 (May, 1934).
Ferguson, Albert B Roentgen Diagnosis of the Extremities and Spine (Annals of Roentgenology, vol. 17), New York, Paul B Hoeber, Inc. 1939.
Keyes, Donald C , and Compere, Edward L. The Normal and Pathological Physiology of the Nucleus Pulposus of the Intervertebral Disc. J. Bone & Joint Surg. 14 897 (Oct , 1932).
Smith, Alan DeForest The Surgical Treatment of Low Back Pain Surgery 4 13-20 (July, 1938)
Willis, Theodore A Low Back Pain - The Anatomical Structure of the Lumbar Region Including Variation J. Bone & Joint Surg. 19 745-748 (1937)
Willis, Theodore A The Separate Neural Arch. J Bone & Joint Surg 13 709-721 (1931)
Willis, Theodore A Anatomical Variations and Roentgenographic Appearance of the Low Back in Relation to Sciatic Pain J. Bone & Joint Surg. 23 410-416 (1941)

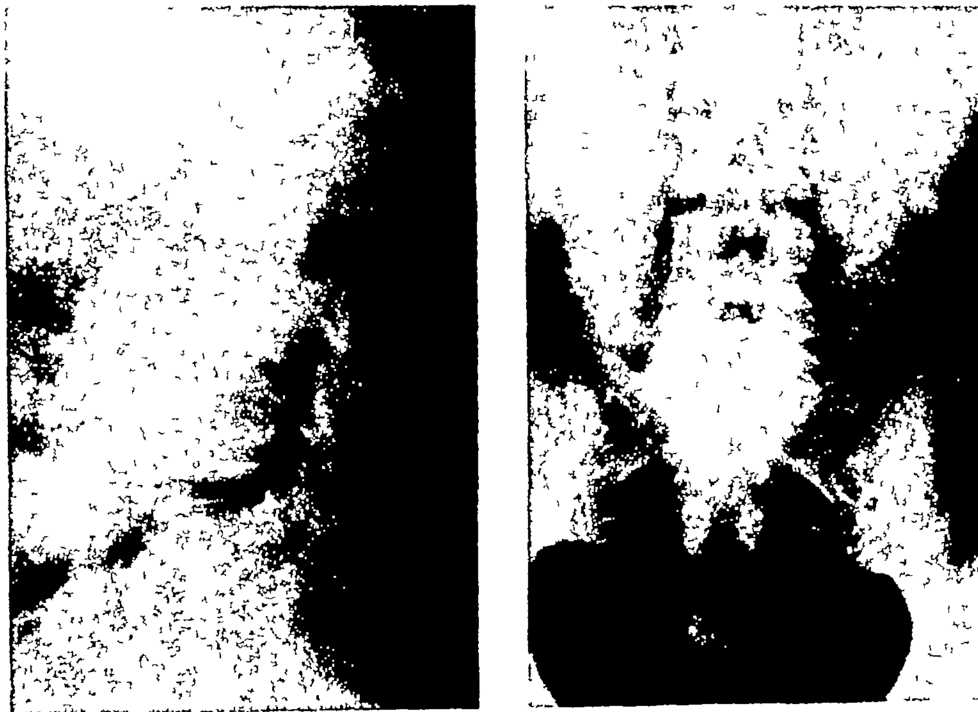


Fig. 1 Lumbosacral fusion for disabling low back pain - Result 9 years after operation. The fusion is solid and the patient has been relieved

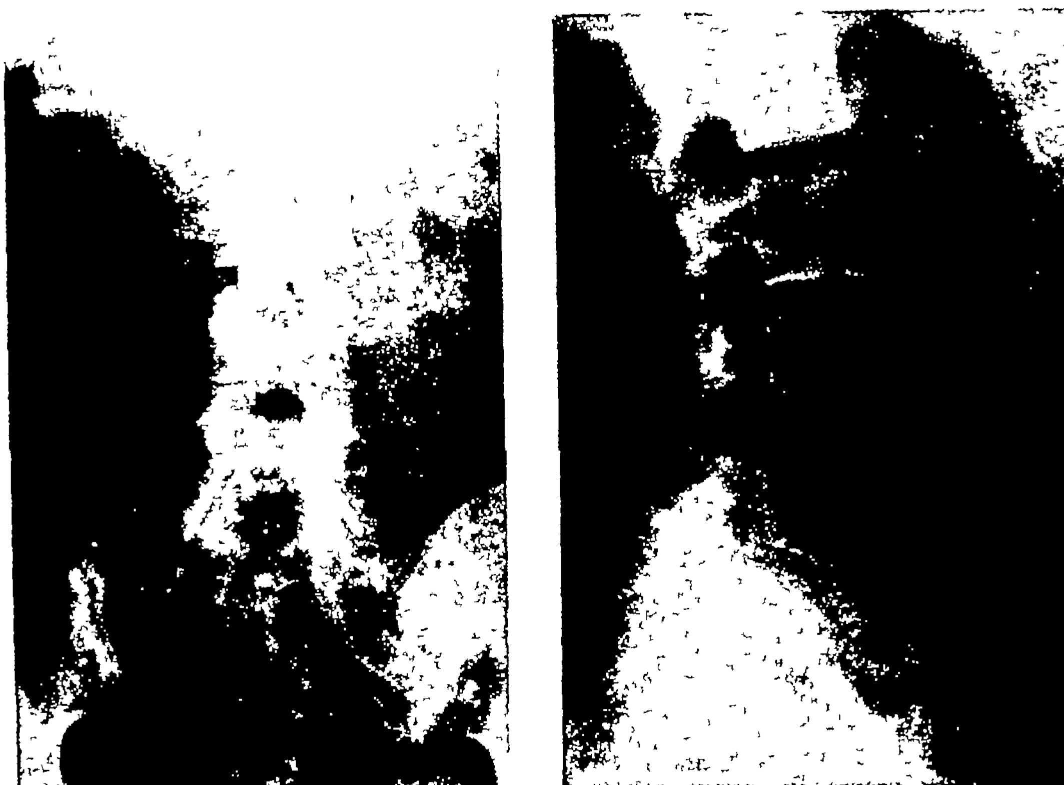


Fig. 2 Internal fixation by screws in lumbosacral fusion - 2 weeks after operation.

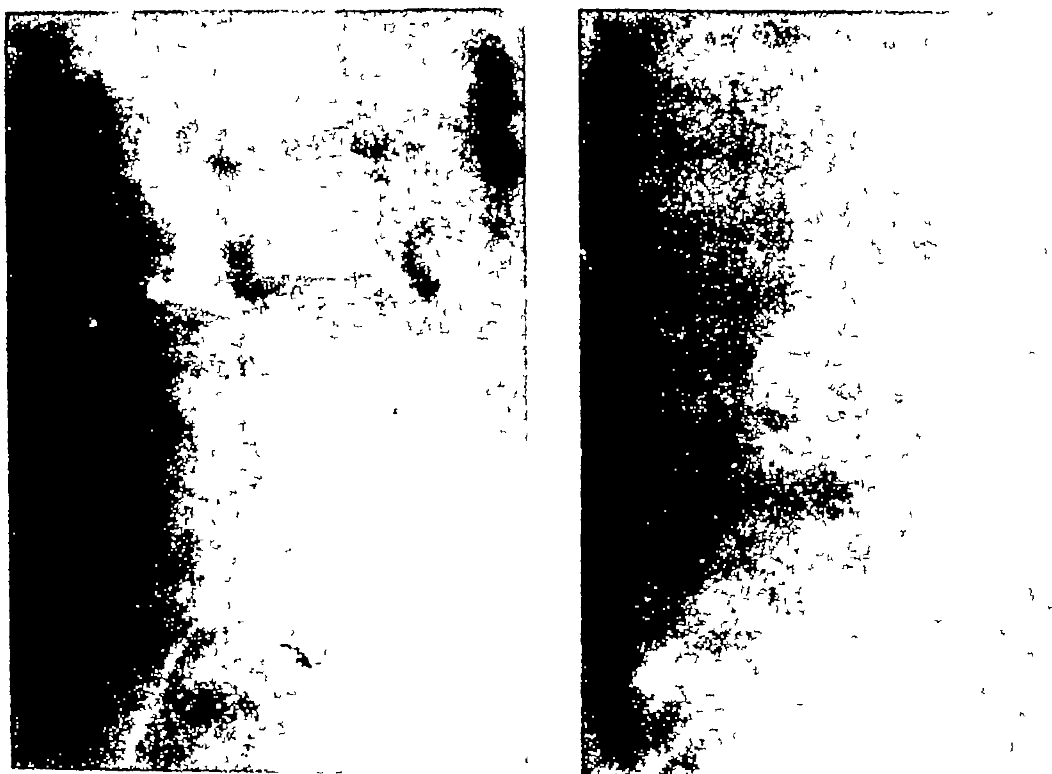


Fig. 3. The use of flexion and extension lateral roentgenograms to detect a pseudarthrosis. The fusion is solid L5 - S1, but a pseudarthrosis permitting motion is present, L4 to L5.

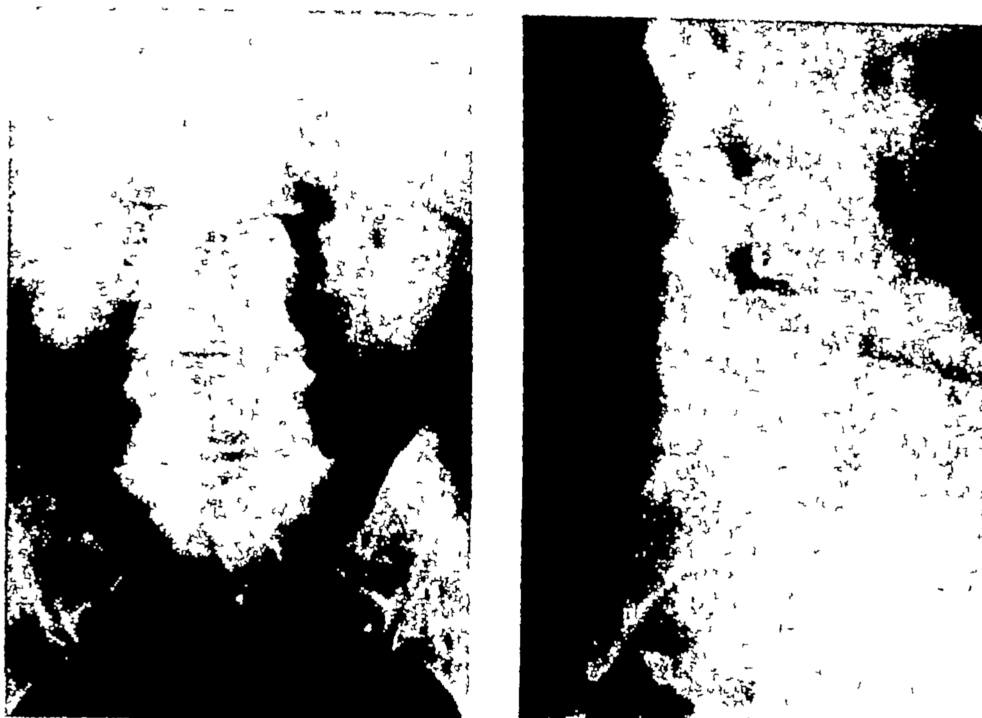


Fig. 4. Repair of the pseudarthrosis L4 - L5 shown in Fig. 3 - 7 months after operation



Fig. 5. Delayed postoperative infection in the intervertebral disc between L4 and L5 following removal of a herniated nucleus pulposus through the interspace. The view on the left was taken three months after operation and failed to disclose any cause for the patient's persistent pain A month later, bone destruction was visible and an abscess of the intervertebral disc was drained Granulation tissue and thin pus were found but the culture was negative. The view on the right shows the lesion three months after the drainage operation The patient has recovered and has been well for several years.

THE DIAGNOSIS AND CONSERVATIVE MANAGEMENT OF LESIONS OF THE LUMBOSACRAL SPINE

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The subject as given is all-inclusive but, for reasons of brevity, infectious and less common lesions of the lumbosacral spine will not be discussed.

Intervertebral disc changes have been diagnosed in 87 per cent of the cases which have entered with the complaint of pain in the lower part of the back. Approximately 5 per cent of this group have been treated by surgical methods, the remainder by conservative measures. This discussion will be confined to the diagnosis and conservative treatment of those presenting intervertebral disc changes.

A cure is not effected, since a potentially painful lesion persists but relief of acute symptoms results and the patient succeeds in learning how to live with the lesion with minimal discomfort.

The conservative treatment of intervertebral disc changes is difficult, as it requires detailed attention and frequent observation of the patient. It is important that the patient understand the mechanical principles involved in order to eliminate the prevalent idea that a military attitude is symbolic of that which is to be desired. An articulated lumbosacral spine has proved to be a most valuable "time saver" in this connection. The articulation of the fifth lumbar vertebra to the sacrum should be accomplished by the use of a sponge rubber intervertebral disc in order to readily permit its collapse and thus demonstrate the subluxation of the lumbosacral facets.

A primary diagnosis of psychoneurosis should rarely be made in these cases, its being more frequently diagnostic of our ignorance than of the patient's primary trouble. As a complication such a diagnosis can be correctly made in many cases.

History

A carefully taken history, in our experience, is just as important in arriving at a correct diagnosis as is the physical examination.

The most common history related is that of an acute onset of pain in the lower part of the back caused by forceful hyperextension. Repeated episodes, usually termed as "lumbago" or "catches," follow for a period of months or years until eventually the attack is accompanied by pain radiating down one of the lower extremities. The most common distribution is related as being down the back and side of the thigh and calf into the lateral aspect of the ankle and foot, thus following the distribution of the fifth lumbar nerve. Less frequently the distribution of pain is described as radiating into the front of the lower thigh and knee and the anterior aspect of the leg, an area which corresponds to the distribution of the fourth lumbar nerve. Still others will describe the distribution of pain as being down the back of the thigh into the back of the calf and heel which corresponds to the distribution of the first sacral nerve. Pain radiating down the inner aspect of the thighs suggests a lesion of the lower urinary tract.

Detailed questioning of those cases who have experienced pain for a considerable length of time will usually reveal the mechanical character of the lesion. The most striking mechanical factor to be learned from the historical data is that extension of the lumbosacral spine increases pain whereas flexion reduces it.

Common related examples are

Sleeping on the abdomen increases pain while sleeping on the side with one or both knees drawn up reduces pain.
Sitting in an erect attitude increases pain but sitting with the knees propped up or with the trunk bent forward approximating the knees reduces pain.
Bending over a wash basin aggravates symptoms but by partially bending the knees and hips the position can be maintained with but little discomfort.
Lifting a load in front of the body at or above the waist line increases pain.
Working with the hands and arms over the head increases pain.
When symptoms are acute, sneezing causes a sharp pain unless done with the knees and chest approximated.

There are numerous other examples to be obtained from the history which aggravates symptoms, such as dancing, prolonged standing, sitting in a theater, rising from a sitting position and driving a car, all of which cause an increase in lumbosacral extension.

Walking is not related as a common cause of aggravation, but when so, it suggests a sacral obliquity due either to a short leg or an asymmetrical development of the sacrum.

Examination

The examination is divided into the following phases: 1) Inspection, 2) palpation, 3) manipulation and motion, 4) measurements, 5) neural examination, 6) impression, 7) x-ray. The patient should be completely nude from behind. A knee-length gown which opens down the back affords such an exposure in the female patient.

Inspection

Inspection should determine the postural type. It should also reveal whether the sacrum is transverse when weight is borne equally on both lower extremities since obliquity of the sacrum must be taken into consideration in treatment. Other changes to be looked for are protective attitudes, exaggerated normal curves, scoliosis, prominent spinous processes, prominence of the sacrum and fibrillary tremors. The so-called sciatic list with its resultant flat or kyphotic lumbar spine seen in acute cases represents an attempt on the part of nature to open the posterior structures of the lumbosacral spine, thus relieving the pressure at this site. The flexion attitude of the involved extremity represents an attempt to relieve tension on the affected nerve Fibrillary muscular tremors usually are the result of an irritation of the nerve supplying the particular muscle involved. A prominent sacrum with a short lumbar spine suggests a spondylolisthesis of the fifth lumbar vertebra with considerable forward displacement. An exaggerated dorsal kyphos in all except certain elderly persons should suggest the deformity of juvenile dorsal epiphysitis, while a sharp kyphos is characteristic of a traumatic or destructive lesion.

Palpation

This is one of the most valuable means of examining a spine. Where there is localized tenderness to jarring or pressure there is usually an underlying lesion. Jarring with the ulnar aspect of the fist will usually determine the presence or absence of a destructive lesion whether it be infectious or a new growth. The complaint of pain as a result of administering a jar over the prominence of the sacrum has been found to be most effective in differentiating intra-pelvic from mechanical lesions. Jarring over a nondestructive lesion of the spine will rarely elicit complaint unless with the mechanical lesion there is an associated meningeal irritation.

Pressure with the thumb will in most cases elicit complaint when applied directly over a mechanical lesion. The exactness with which such a lesion can be localized with this most simple test should be further emphasized. Light rotatory pressure with the distal palmar surfaces of the fingers over the gluteal region will locate painful fibrolipomata, an infrequent but definite clinical entity occurring usually in hypogonadal and menopausal women. Tenderness to pressure over the femoral or sciatic nerves suggests an irritative lesion of the nerve or one of its component segments. The degree of muscle spasm and tautness of the tensor fasciae can be determined by palpation.

Manipulation and Motion

Variations from the normal in the ranges of motions of the lower spine and the hips should be determined. A spine that moves as one piece is characteristic of a Marie-Strumpell's disease or a far advanced rheumatoid arthritis. A severe hypertrophic arthritis is occasionally responsible for this finding. When the lumbosacral spine presents limitation and painful anterior flexion, with deviation to one side accompanied by limitation and painful extension, and occasionally limitation and painful lateral flexion to the affected side, it is indicative of a collapsed lumbosacral intervertebral disc causing acute symptoms.

Motions of the hip joints are unaffected in lesions of the lower spine except as the patient resists motions in an effort to protect his spine The examiner must be able to differentiate such protection from true limitation of the hips. The latter is indicative of a lesion of the hip joint. The lesion is usually intra-capsular if motions are limited in all directions but extra-capsular if limited in less than all

directions.

There are numerous manipulative tests which must be included within this phase of the examination. Lasègue's sign produced by the straight leg raising test when present indicates a neuralgia of the sciatic nerve or spasm of the hamstring muscles. The examiner should differentiate between hamstring spasm and hamstring limitation due to a loss of lumbosacral flexion resulting from a long continued mal-posture.

The straight leg raising test will not afford differential diagnostic information as regards a sacro-iliac and lumbosacral lesion. It is the author's experience that the sacro-iliac joint causes clinical symptoms far less frequently than does the lumbosacral joint and that in those cases where it is causing symptoms there is an associated collapse of the lumbosacral intervertebral disc. The force that most frequently causes a strain of the sacro-iliac joint is the same force that causes a compression of the posterior aspect of the lumbosacral intervertebral disc. The intervertebral disc, being more susceptible to trauma, collapses before the strain of the sacro-iliac joint becomes of clinical significance. A true clinical picture of a sacro-iliac strain can best be evaluated in those who have previously had a lumbosacral fusion. Five such cases have been studied. The most common history related was that of a sudden and severe onset of pain in the posterior hip region while lifting a load in front of the body. The usual findings have been a localization of major tenderness over the inferior border of the sacro-iliac joint, a moderate limitation of anterior flexion of the trunk at the hip joints with an accompanying complaint of pain referred to the posterior upper thigh and a positive Lasègue's sign due to spasm of the hamstring muscles. The passively forced extremes of any and all motions of the hip on the affected side caused a complaint of pain referred to the sacro-iliac region. In all cases there was an absence of protective spinal attitudes as well as segmental nerve symptoms.

Patrick described what he called the faber test for diagnosing lesions of the hip joint. Its use in examining the patients under consideration has become quite popular. As a means for which it was intended it remains a valuable test, however, there has developed a prevalent opinion that when positive it is diagnostic of a sacro-iliac lesion. There apparently is no unanimity of opinion as to what constitutes a positive test in this connection. Aside from the purpose for which it was intended the test has a value in revealing the presence of a femoral nerve neuralgia just as the straight leg raising test is of value in obtaining the same information about the sciatic nerve.

The Ober test can be used in determining the tautness of the fascia lata, however, the same information can be obtained by less complicated manipulations. Our concern in the fascia lata is to determine whether its tautness is of such a degree that it is binding down the front of the pelvis and thus maintaining a persistent hyperextension of the lumbosacral spine.

The most simple yet accurate method of determining the tautness of these structures is by palpation alone, done with the patient standing. The Thomas test is not only an excellent method of determining a flexion deformity of the hip but it also affords an efficient means of estimating the degree of tautness of the fascia lata. Its use is employed in all suspected lesions of the lumbosacral spine.

There are other less popular tests which occasionally can be used to a good advantage in certain cases, but those already mentioned are undoubtedly the most satisfactory and therefore more generally employed.

Measurements

Any variation in the length of the lower extremities should be determined. The customary procedure of determining such is to measure the distances from the anterior-superior iliac spines to the medial malleoli after it has been determined that the pelvis is in a transverse plane and that the lower extremities are in the same relative position with the pelvis This procedure will reveal in most cases a variation in the length of the lower extremities, however, it fails to furnish the desired information in many cases. The reason for acquiring this information is to determine whether the weight of the trunk is resting on an oblique sacral table. A developmental disturbance of the sacrum in which one side is shorter than the other is not infrequently seen and causes a sacral obliquity. The resultant lateral tilt of the sacral table affects weight bearing at the lumbosacral articulation the same as does a short leg. A more accurate

determination of a sacral obliquity can be determined by inspection alone, as already mentioned.

Circumferential measurements of thighs and calves should be made where inspection has revealed a variation in appearance. An atrophy of one of the lower extremities suggests a neuritis of one of the lumbar or sacral nerve segments although it can result from any lesion of the extremity which causes the patient to favor the part.

Neural Examination

A neural examination of value requires on the part of the examiner a thorough knowledge of the innervation of the musculature and of the topography of the dermatomes of the lower extremities. The classical work of Foerster[1] on the dermatomes of man must be accepted as authoritative. His findings corresponded fairly accurately with those previously submitted by Head. The changes in the dermatome topography of the lower extremities recently suggested by Keegan[2] lack scientific substantiation. The works of Foerster has shown that there are individual variations and marked overlapping of the various dermatomes.

A variation in the sensation of that area supplied by the fifth lumbar nerve whether it be hyperaesthesia, hypaesthesia, or anesthesia suggests a collapse of the fifth lumbar intervertebral disc with compression of the nerve roots at the foramina. This finding may also be present in a ruptured fourth lumbar intervertebral disc providing the prolapse of the nucleus is of such magnitude that it compresses the fifth lumbar nerve segment. A sensory alteration of that area supplied by the fourth lumbar nerve suggests a collapsed fourth lumbar intervertebral disc with irritation of the fourth lumbar nerve root at the foramen. A similar nerve change may be observed in cases presenting a ruptured third lumbar intervertebral disc, providing the protruding nucleus is of such magnitude that it compresses the fourth lumbar nerve segment. A sensory alteration in the area supplied by the first sacral nerve suggests the presence of an extra lumbar vertebra with a collapse of the lumbosacral intervertebral disc or a partial lumbarization of the first sacral vertebra or a collapse of the fifth lumbar intervertebral disc with posterior protrusion of such a magnitude that it compresses the first sacral nerve root

The fifth lumbar intervertebral disc, being more subject to both acute and chronic trauma, undergoes collapse more frequently than do other lumbar intervertebral discs. The segmental changes associated are usually confined to the fifth lumbar nerve. Since this nerve segment emerges from the neural canal without contacting the fifth lumbar intervertebral disc it is logical to conclude that its irritation is occurring at the constricted foramen.

New growths of both intra- and extradural origin should be considered in all cases. A test for Babinski's reflex and ankle clonus should be made routinely in order to rule out an upper motor neuron lesion

An alteration in the Achilles reflex is a common finding. The reflex may be hyperactive, diminished, or absent. The hyperactive state is usually observed in those cases experiencing early mild irritation. The diminished or absent reflex is characteristic of a more severe and prolonged compression. An alteration of this reflex is most commonly observed in a fifth lumbar nerve neuritis although it undoubtedly can be associated with a first sacral nerve neuritis. An alteration in the knee reflex is not infrequently observed. Here, as with the Achilles reflex, its action may be increased, diminished, or lost. Its change is indicative of a neuritis of one of the segmental components of the femoral nerve, usually the third or fourth lumbar. An alteration in the adductor reflex suggests a neuritis of the second lumbar nerve.

Impression

From the physical findings alone one can readily gain the impression that the patient is suffering from a lesion of the lumbosacral spine. Its location can be fairly accurately determined, especially if segmental symptoms are presented, however, the inability to demonstrate pathological changes by x-ray has caused extreme confusion within the mind of the physician. The inability to demonstrate such changes has been due principally to improper posing of the patient at the time the films were made and to a failure to interpret properly the changes which are presented

X-rays

The bone changes associated with a collapsed intervertebral disc are detailed in

character and can readily be hidden from view by improper posing of the patient. The individual variation in the bone architecture of the lumbosacral spine varies to such an extent that mathematical rules regarding angles cannot be satisfactorily employed.

Five films of the lumbosacral articulation are routinely taken on all patients. The first consists of a lateral spot centered over the lumbosacral articulation. Formerly this film was taken with the patient in a weight-bearing position, but we have found that the technical problem is simplified in the nonweight-bearing attitude. Providing certain principles are followed, the difference as recorded by the film is inappreciable. In the reclining position the hips must be in extension and the spine so supported that it is parallel to the table. The extension of the hips should be equivalent to that of the weight-bearing position. The lateral film is developed and interpreted before proceeding with the other exposures. The degree of tilt of the lumbosacral table is determined from the lateral film. With this information the necessary angle of the central ray is determined in order that the latter may pass through the lumbosacral space while making the anteroposterior exposure. A stereoscopic anteroposterior study is made. A lateral rather than a vertical shift is preferable in making this study in order that the central ray may pass unobstructed through the lumbosacral intervertebral space in both positions.

Single oblique studies of the lumbosacral facets are then made. A tilt in the central ray is just as important in making the exposures for the oblique studies as it is in making those for the anteroposterior exposures. Otherwise a distorted study from superimposed bony structures will result. The tilt of the central ray should vary with the individual inclination of the sacral table just as in the anteroposterior exposure. Fig. 1 is that of an oblique film taken with the central ray tilted as described. Fig. 2 shows an oblique study taken with the central ray in a vertical position.

Providing good equipment has been employed the five films mentioned usually show changes sufficient to make a correct diagnosis.

The final interpretation of the films is the responsibility of the orthopedist rather than the roentgenologist, however, the controversy is usually insignificant once the roentgenologist becomes familiar with the changes with which the orthopedist is concerned.

Since the intervertebral disc does not cast a shadow on the x-ray films, an interpretation must be based primarily on secondary bony changes. The principal secondary changes which follow a destruction of the intervertebral support are a subluxation of the adjacent facets as shown in Fig. 1, posterior displacement of the segment immediately above the involved disc as shown in Fig. 7, narrowing of the intervertebral space as shown in Fig. 6; contact of adjacent spinous processes as shown



Fig. 1. Oblique view of lumbosacral facets showing true subluxation of facets as a result of tilting the central ray.



Fig. 2. Oblique view of lumbosacral facets showing distortion caused by the central ray being directed in the vertical position



Fig 3 Lateral view showing findings characteristic of a collapse of both the fourth and fifth lumbar discs Further settling of the fifth lumbar segment is prevented by lodging of the first sacral facets Further settling of the fourth lumbar segment is prevented by contact of the fourth and fifth lumbar spinous processes.

in Fig. 3, marginal lipping on adjacent vertebral surfaces caused by abnormal stress resulting from loss of the resilient intervertebral support (Fig. 5). Lodging of the superior margins of the first sacral facets in the inferior vertebral notches of the fifth lumbar vertebra thus transforming the superior first sacral facets into weight-bearing structures and causing sclerosis of the latter as well as sclerosis within the inferior vertebral notches and occasionally osteophyte formation at the latter site (Fig. 4 and Fig 5). In turn this usually results in an osteoporosis of disuse in the inferior articular facets of the fifth lumbar vertebra. Another change occasionally observed is the "saddle effect" as described by Badgley[3], which is the result of the inferior facets settling in the sulci of the laminae of the segment immediately below

There are other less frequent secondary changes which are apparent by x-ray, but those mentioned include the important changes. The subluxation of the facets is the most important diagnostic change. Myelograms are used in those cases which present a fixed resistant clinical picture after thorough conservative treatment and in those cases suggesting a cord lesion at the time of the original examination.

The narrowed intervertebral space when properly interpreted adds valuable diagnostic information Whether the entire space is narrowed or just the posterior portion depends on the acuteness of the lumbosacral angle and the magnitude of the first sacral facets. It has been interesting to note that the presence or absence of segmental symptoms depends to a large extent upon these same factors. Fig. 6 shows a narrowing of the entire lumbosacral intervertebral space. It also shows a good lumbosacral angle with small superior first sacral facets which are not lodged in the inferior vertebral notches of the fifth lumbar vertebra. Thus, the fifth lumbar nerve roots are not impinged even though the foramina are somewhat reduced in size. This particular case lacked segmental symptoms

Fig. 7 shows a lumbosacral space which would rarely be interpreted as being narrowed,

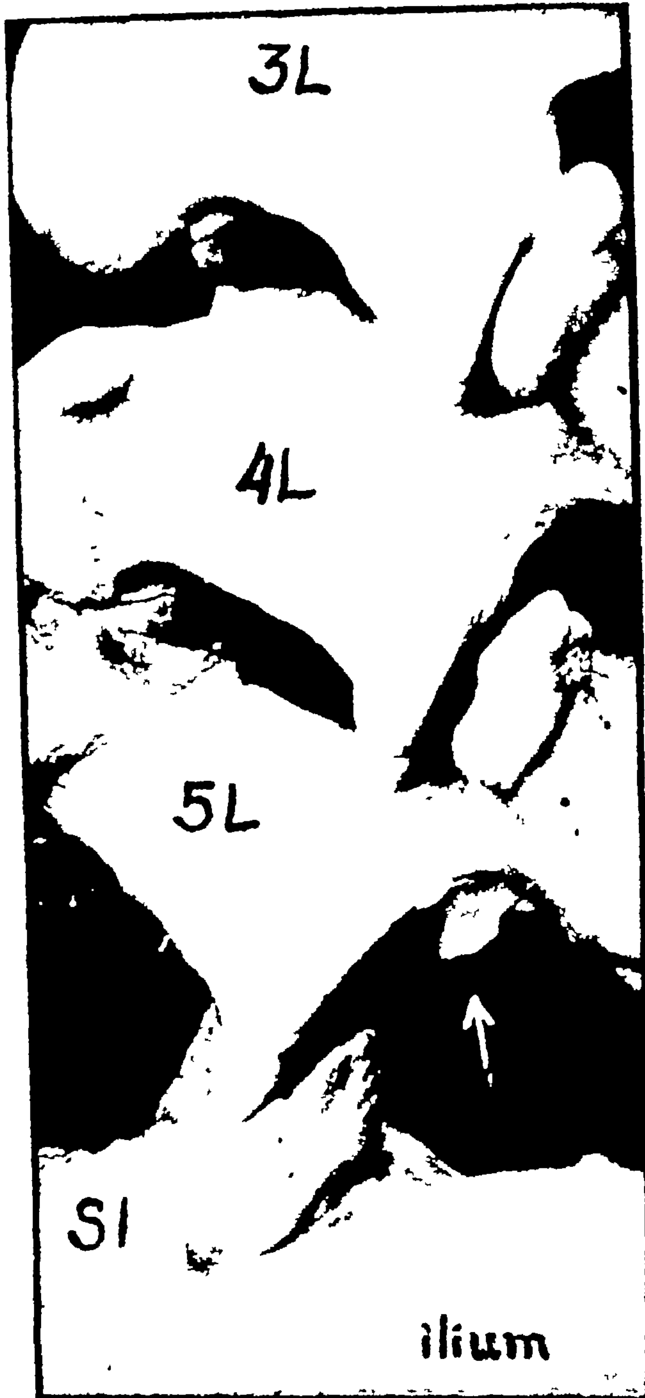


Fig 4. Skeletal material showing the result of first sacral facet lodging in inferior vertebral notch. Note sclerotic appearance of sacral facet and osteophyte formation within the inferior vertebral notch.

however, it shows an extremely acute lumbosacral angle with very large first sacral facets resting in the inferior vertebral notches, thus preventing any further narrowing of the lumbosacral intervertebral space. Other films of this same case show extreme subluxation of



Fig 5. Oblique view of lumbosacral spine showing osteophyte formation within the inferior vertebral notch of the fifth lumbar vertebra. Also narrowing of the fourth lumbar vertebral space with marginal lipping on adjacent vertebral surfaces and subluxation of facets.

the lumbosacral facets. This case presented a severe fifth lumbar nerve neuritis.

Treatment

The lesion under consideration is mechanical in character and therefore must be treated along mechanical lines. An understanding of the pathological changes and the physiology of the part is essential in establishing correct mechanical principles of treatment. Fig. 8 shows the anatomical relationship and some of the changes which accompany a collapse of the lumbosacral intervertebral disc. The superior margin of the first sacral facet is shown to be in contact with the inferior vertebral notch. The anterior superior aspect of the facet is contacting the funicular portion of the fifth lumbar nerve. The prolapse of the annulus fibrosis and the subluxation of the joint are shown. It becomes apparent from a study of the specimen that contraction of the erector spinae at a time when the lumbosacral joint is in an extended position will tend to elevate the sacrum which in turn forces the anterior superior aspect of the first sacral facets into firm contact with the fifth lumbar nerves within the foramina. It is under such conditions



Fig. 6 A complete loss of the joint space follows a destruction of the lumbosacral disc when the lumbosacral angle is not acute and the superior first sacral facets are not large

that most acute attacks of pain occur, for example when lifting a load above the waist line, raising a resistant window, pivoting on a golf shot, or arising from a sitting position

The erector spinae and the hip flexors are the most important extensors of the lumbosacral spine. The anterior abdominals and the glutei maximi are the most important flexors of the lumbosacral spine Treatment should be directed at reducing lumbosacral extension and thereby relieving the posterior pressure at this site. It is therefore necessary to actively develop the flexors of the lumbosacral spine and passively stretch the extensors in order to obtain and maintain a satisfactory postural balance and thus reduce lumbosacral extension.

The symptoms from a collapsed lumbosacral intervertebral disc may be acute, chronic, or quiescent. The treatment must be instituted accordingly.

It is the opinion of the writer from incomplete studies that more than 60 per cent of adult population have a collapse of the lumbosacral intervertebral disc and thus a potentially painful lesion.

The physician rarely has an opportunity to treat those who have a lesion in the quiescent stage except when encountered accidentally or unless they report with the complaint of "poor posture." When such is the case they should be treated the same as those suffering with chronic symptoms.

Chronic Stage

Those suffering chronic symptoms constitute the majority of "low back" patients who report for treatment. In this group the aggravation of symptoms by bending over a wash basin, rising from a sitting position, prolonged standing, and sleeping on the abdomen are common complaints. Treatment consists of the institution of a postural program If the patients present a sacral obliquity it should be compensated for by a lift on the heel of the shoe unless pain radiates down the short extremity If such is the case, a compensatory lift will usually aggravate segmental symptoms. If pain radiates down the long extremity the compensatory lift usually affords spectacular relief. After correcting lumbosacral weight bearing in the lateral plane, efforts are then directed at its correction in the antero-posterior plane - that is, a reduction of the lumbosacral angle.

An exercise program directed at actively developing the flexor muscles of the lumbosacral spine and passively stretching the extensor muscles is instituted. Fig. 9 shows the exercises which are employed. They should be taken on a padded floor and the group should be taken twice daily at a minimum. The number of times each exercise should be taken must vary with the individual case The variation has been from 10 to 50 times during our experience. The abdominal exercise is as a rule the most strenuous and therefore its execution is broken by alternating with the others. Exercise 1, Fig. 9, is directed at actively developing the anterior abdominal muscles. The patient should not anchor the feet, because in so doing the hip flexors are thrown into active contraction. Whether a patient can rise to a full sitting position or only part way does not depend on muscular strength but rather on the relative weight of the trunk and the lower



Fig. 7. Severe lumbosacral angle and large first sacral facets retains width of lumbosacral space. First sacral facets are resting in inferior vertebral notches preventing further narrowing Fifth lumbar vertebral body is posteriorly displaced.

extremities, the buttocks acting as a fulcrum.

Exercise 2, Fig. 9, is directed at actively developing the glutei maximi. The pelvis is rotated forward by an active contraction of these muscles. The hands are placed on the abdomen just above the umbilicus and should hold the upper abdomen down so that flexion takes place only at the lumbosacral spine.

Exercise 3, Fig. 9, is directed at passively stretching the erector spinae and at the same time restoring flexion to the lumbosacral spine. The grip on the knees should not be released during the course of the exercise. The thighs should be spread apart in order to avoid contact with the abdomen or chest. The knees are then pulled back and forth in an effort to bring them into the axillae. Avoid trying to pull the knees above the shoulders since this results in flexion of the upper dorsal and cervical rather than the lumbosacral spine.

Exercise 4, Fig. 9, is intended for stretching the fascia lata, which is not necessary in all cases. Its indication is determined by physical examination. When the fascia lata are even moderately taut its use is important and will be found to be quite effective when practised over a period of time. The foot on the flexed extremity should be placed and remain flat on the floor while the foot on the extended extremity should be dorsiflexed so that weight is borne on the ball of the foot rather than on the tips of the toes. By flexing the forward knee the pelvis is moved up and down, the downward motion being effective in stretching the taut structures.

Exercise 5, Fig. 9, is directed at restoring lumbosacral flexion and stretching the erector spinae. Still more important is its action in stretching short hamstring muscles in those cases who have experienced protective hamstring spasm over a long period of time. This exercise is not indicated in all cases but is most effective in those resistant cases already mentioned.

The "flat footed squat" which has been emphasized by Regen has been found to be very effective and is employed in the conservative treatment of most cases. It is an exercise which can readily be used at intervals throughout the day. It is executed by placing the feet parallel and about fifteen inches apart. The patient then squats with the back rounded until the buttocks are about eight inches from the floor. This should be accomplished without raising the heels from the floor. He is advised to squat in this manner at least five times and to repeat the process six to eight times daily. In middle and late life many find it impossible to do this exercise as outlined,



Fig. 8. Anatomical specimen showing subluxation of the lumbosacral facets and impingement of the fifth lumbar nerve by the first sacral facet. (Courtesy of Dr. Yglesias)

but through persistent effort they can usually accomplish it over a period of a few weeks. Such persons should use a weight of six to eight pounds held out in front of their trunks to act as a counter-balance. Still others are so disabled that they are instructed to start by attempting to arise from a sitting position on a stool about eight inches high, if necessary by using a weight as a counter-balance.

The exercises as outlined should not be altered except at the direction of a physician and then only after considerable thought has been given to the keniseological action involved. As an example, Exercise 5 could be taken in a standing position, but if so done it would result in active development of the erector spinae muscles which in civilized man are already overdeveloped and to a large extent responsible for symptoms.

The exercises are essential in obtaining proper muscular balance and restoring flexion to the lumbosacral spine, but good posture is acquired only through conscious effort. The patient is taught to live 24 hours a day with extension of the lumbosacral spine reduced to a minimum. This requires instructions in standing, walking, bending, sitting and reclining.

Standing and walking should be done in a position of "forward attack." The chest should be the part farthest forward of the trunk. The front of the pelvis should be lifted so that it approximates the xyphoid cartilage, thus producing a crease across the upper abdomen. The feet should be pointed straight ahead and most of the body weight should be borne on the

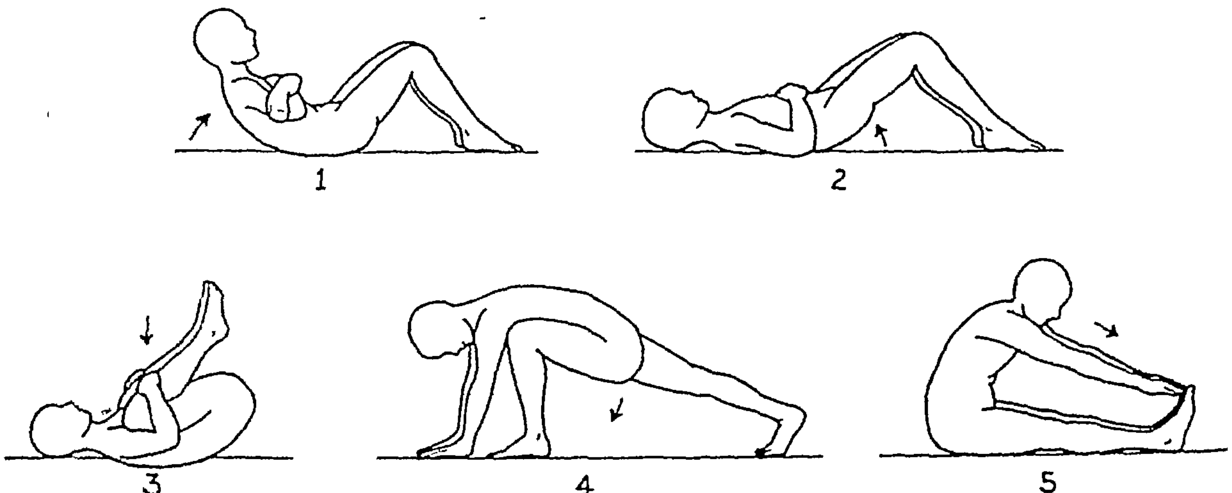


Fig 9 Postural exercises.

heels. To carry the weight on the balls of the feet causes a forward shift in the base of support which is compensated by a forward thrust of the pelvis and a backward thrust of the upper trunk with a resultant increases in lumbosacral extension. The use of high heels should be avoided as much as possible since any elevation of the heels shifts the base of support forward with a resultant compensation in the form of lumbosacral extension.

When in the erect attitude patients are taught to get up and down by first flexing the lumbosacral spine and then "squatting." The latter should consist of acute flexion of both the hips and knees. A load should not be lifted with the knees in extension as shown in Fig. 13 G. Instead it should be lifted with the legs and with the lumbosacral spine in flexion as shown in Fig. 13A. A load should not be carried in front of the body at the level of or above the waist line as shown in Fig. 13H except with the lumbosacral spine rounded and the hips and knees partially flexed, as in Fig. 13B. A load should not be lifted above the waist line because of the necessary increase in lumbosacral extension and the increased force on the posterior structures at this site.

Sitting should be done with the buttocks "tucked under," thus flexing the lumbosacral spine (Fig. 13D). It is advisable to elevate the knee joints above the hips in order to further flex the lumbosacral spine. Most chairs are too high for the average woman, causing her to sit with the knees at a level lower than the hips and thus extending the lumbosacral spine as shown in Fig. 13J. It is customary and commendable for women to partially overcome this faulty position by crossing the knees (Fig. 13E), but a stool is usually necessary if comfort is to be maintained in prolonged sitting on such a chair (Fig. 13F).

A hasty glance at any male audience will readily reveal the urgent tendency to acquire lumbosacral flexion and with it comfort.

A correct driving position is of special importance to those whose occupation demands this mode of travel. The tendency on the part

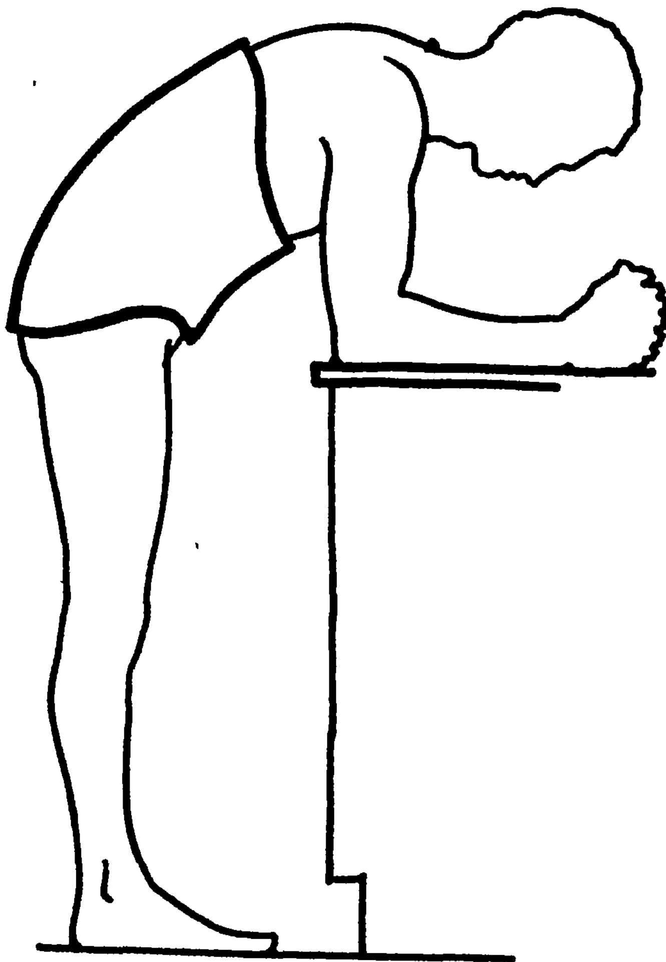


Fig 10. Flexion plaster of Paris jacket.

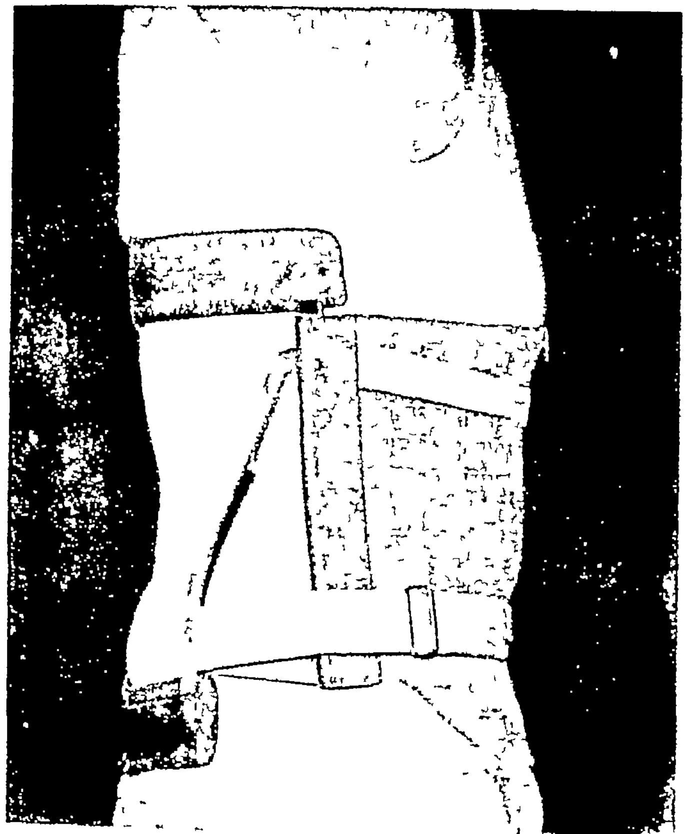


Fig. 11. Lateral view of lumbosacral flexion brace



Fig. 12 Flexion attitude employed in relieving acute cases. A similar sleeping attitude is advised for those who suffer at night.

of the male is to push the driver's seat back, thereby lowering the knees and increasing the lumbosacral angle as illustrated in Fig. 13P. On the contrary the driver's seat should be pushed forward, thereby raising the knees to a level higher than the hips and thus tending to reduce lumbosacral extension as illustrated in Fig. 13O. The frequent complaint of inability to sit through a show is due largely to the inclination of the theater floor which requires that the knees come to rest at a plane below that of the hips, thus increasing lumbosacral extension as shown in Fig. 13K.

Sleeping on the abdomen increases the lumbosacral angle and is frequently related as cause of increased pain (Fig. 13M). Such a sleeping attitude should be avoided and the patient taught to sleep on the side with one or both knees drawn high enough to flex the lumbosacral spine as shown in Fig. 13L. The bed should be firm. Many patients of their own accord acquire this position in their constant search for comfort. Sleeping on the back with the knees elevated is recommended as the most effective sleeping position. It is preferable to obtain elevation of the knees by a roll under the mattress as shown in Fig. 13N, rather than using pillows which so readily become displaced. Should the patient turn on his side in his sleep because of the elevation in the bed, he will unconsciously draw up one or both knees, thus maintaining flexion of the lumbosacral spine. The most efficient flexed position of sleeping is obtained by the use of a hospital bed as shown in Fig. 12. Its permanent use is recommended in many chronic cases especially in elderly persons where mechanical means of correction have to be employed more frequently.

Mechanical support is reserved for those cases who lack ability to obtain or maintain satisfactory postural positions, many elderly patients fall into this group. Corsets are rarely advised because they prevent the assuming of correct postural attitudes and lack corrective qualities. The relief frequently obtained from their use is due primarily to immobilization rather than correction. When a mechanical support is indicated a brace of the three-point pressure principle designed by the writer[4] and previously described is employed. A lateral view of such a brace is shown in Fig. 11.

The flexion plaster-of-paris jacket can be used to advantage in certain chronic cases who are unable to habituate themselves with correct postural principles. The cast should not be used until the patient has restored practically full flexion to the lumbosacral spine. It is applied with the patient standing, bent forward with his elbows resting on a table at approximately waist-line height, as shown in Fig. 10. After application it is cut vertically in front, removed, and dried. The cut edges

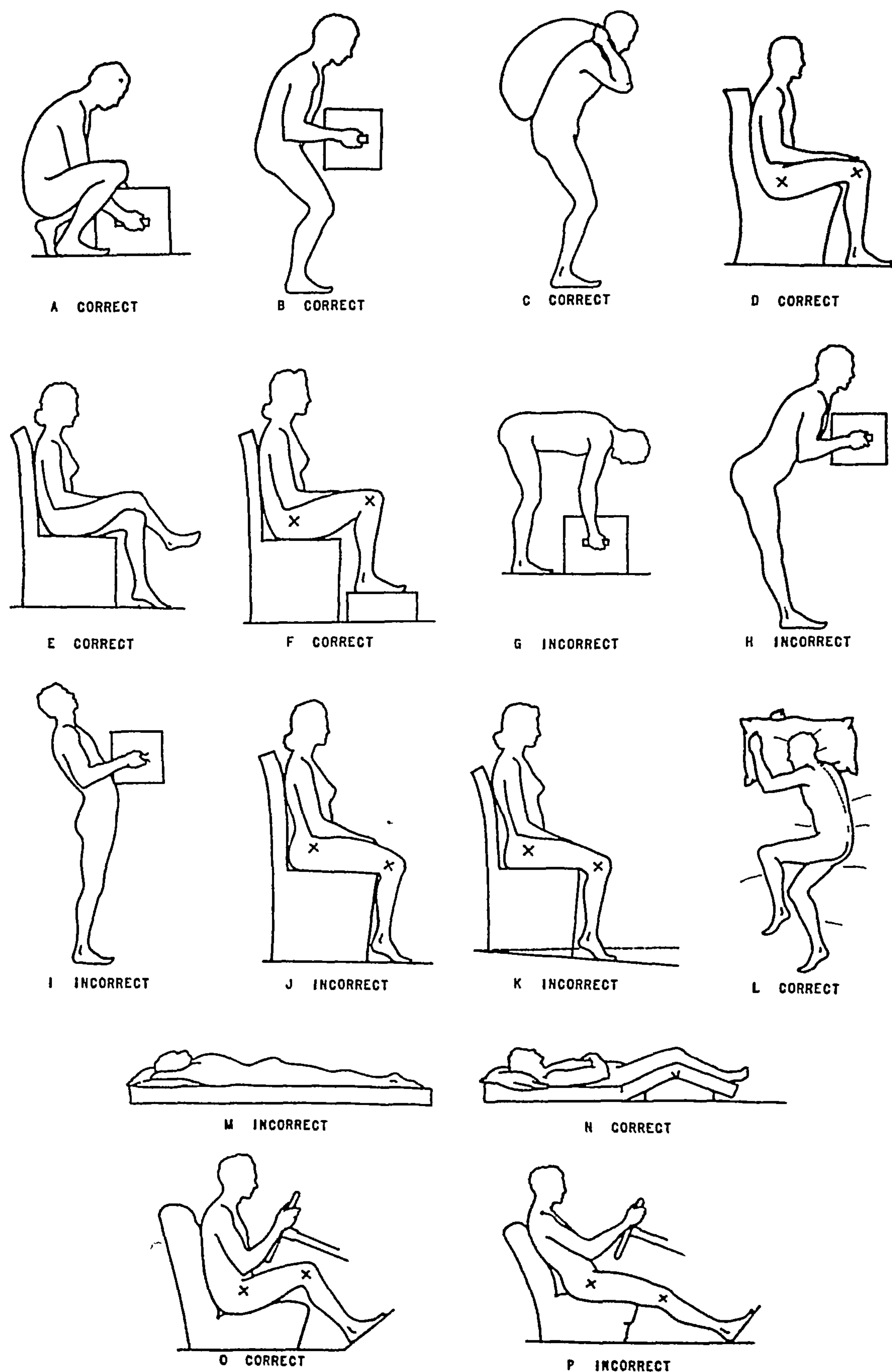


Fig 13. Correct and incorrect postural attitudes.

are then bound, and it is reapplied and pulled together by means of a belt The patient is instructed to remove it at night and to take his exercises after removal and before reapplying in the morning It is worn from two to six weeks as indicated in the particular case

Acute Stage

Acute cases are admitted to the hospital and placed in a position of flexion as shown in Fig 12. Eight to ten pounds of skin traction is applied to the painful extremity in those cases who have acquired a flat or kyphotic lumbar spine as a result of a protective spastic attitude.

Flexion is considered of more importance in treatment than traction and therefore is maintained at all times. Medication consists of the regular administration of an antispasmodic and a narcotic for the first 48 hours. Thereafter, their use is altered as indicated by the progress of the patient. After traction has been applied the patient is instructed in Exercises 1 and 2 to be taken in bed at the end of every hour when awake The number of times each exercise is executed varies with the individual case but the usual starting number is ten The number is increased daily as tolerated. The traction is allowed to remain 4 to 7 days after which it is removed and Exercise 3 is added to the program. The length of time required to relieve acute symptoms depends on the severity of the case but the average patient is ready for discharge after a period of 7 to 10 days When acute symptoms have subsided a complete postural program is instituted and the patient is treated the same as the chronic cases. Frequently when recent symptoms have been severe a plaster-of-paris flexion jacket is applied before patient becomes ambulatory in order to avoid exacerbation during the remaining convalescent period

All cases should be checked for possible foci of infection as these may play an important secondary role in the aggravation of symptoms in those cases which present a lesion of the lumbosacral spine. Removal of a frank focus of infection will frequently result in relief of clinical symptoms, however, there still remains a potentially painful lesion in the lumbosacral spine which should not be disregarded.

Occasionally a fasciotomy as described by Ober[5] can be employed to good advantage in those cases which present a marked tautness of the fascia lata and who fail to respond satisfactorily to a conservative program. This procedure should not be heralded as a cure but only as a further aid in the program of reduction of lumbosacral extension.

Manipulations, injections, and physiotherapeutic measures too numerous to mention have been described. The value in all such procedures is that of interrupting the muscle spasm in acute cases. Many of these procedures are of value in this respect and are employed in connection with flexion principles but it is our opinion that they have nothing permanent to offer as regards future progress of the individual case.

One of our greatest needs within the specialty of orthopedic surgery is a standardization of postural exercises. Many exercises employed and classed as postural exercises are actually defeating the purpose for which they are intended and thus increasing symptoms. An example of such is the straight leg raising exercise which was so commonly employed in the Army and Navy programs and which is used in some of our best institutions as a means of developing the abdominal muscles. Within the first 30 degrees of elevation of the legs the psoas muscles are called into violent action Their action on the low spine at this stage of the exercise is one of compression and extension of the lumbosacral spine due to the location of their origin which lies posterior to the vertical axis. This exercise frequently initiates symptoms in those who have not previously experienced pain in the lower part of the back.

REFERENCES

1 Foerster, O The Dermatome in Man Brain 56 690-703 (1933)
2 Keegan, J Jay Neurosurgical Interpretation of Dermatomes Hypalgesia with Herniation of the Lumbar Disc J Bone & Joint Surg 26 238-248 (1944)
3 Badgley, Carl E The Articular Facets in Relation to Low Back Pain and Sciatic Radiation J Bone & Joint Surg 23 481-496 (1941)
4 Williams, Paul C Lesions of the Lumbosacral Spine, Part 2 J Bone & Joint Surg 19 343-363 (1937)
5 Ober, F R Back Strain and Sciatica J A M A 104 1580 (1935)

Course No. 7

CONGENITAL DISLOCATION OF THE HIP

Lecturers

Joseph A. Freiberg, M.D , Etiology, Pathology and Diagnosis of Congenital Dislocations of the Hip

H. R. McCarroll, M.D., Early Management of Congenital Dislocation of the Hip

A. Bruce Gill, M.D., The Operative Treatment of Congenital Dislocation of the Hip· Indications and Methods

ETIOLOGY, PATHOLOGY, AND DIAGNOSIS OF CONGENITAL DISLOCATION OF THE HIP

Joseph A. Freiberg, M.D.

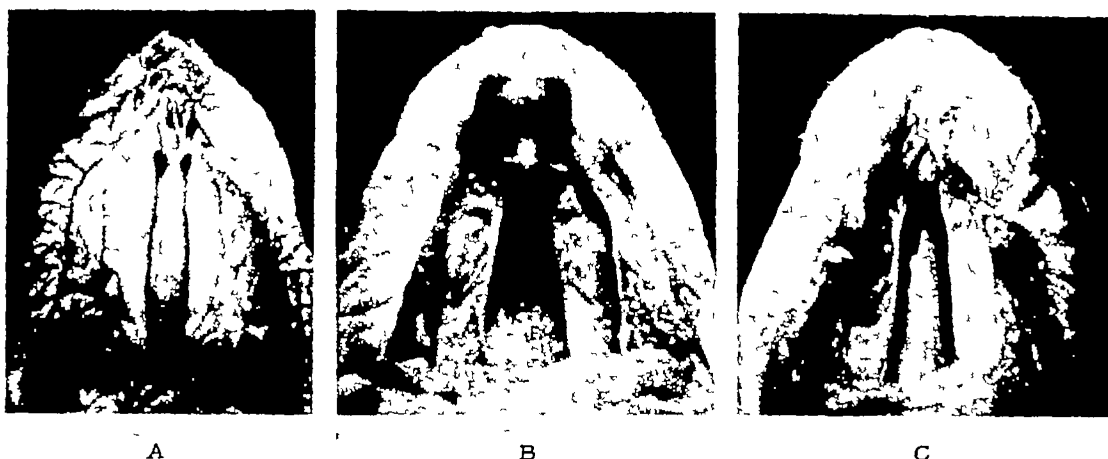
Statistics

THE UNITED STATES Crippled Children's Bureau had on state registers on December 31, 1944, a total of 321,022 crippled children, of which the total number of congenital dislocations of the hip were 6,795, or approximately 2%. The total reported from the several states varied, of course. Some states included cardiac cases, others did not. In general fewer cases were reported in the southern states. The largest per cent of the total represents polios. In foreign countries the figures for congenital luxations were much higher (Italy, Austria, Germany, Czechoslovakia). The figures from Italy (Scaglietti[1]), for example, showed that of 4379 congenital deformities (1899-1932) 3216, or 73%, were congenital hip luxations, 787, or 18%, were club feet, and 116, or 2.65%, were torticollis. As cardiacs and polios are not included, the statistics are not comparable, but nevertheless the much larger number of congenital hips is readily evident. Both Isigkeit,[2] from Saxony, and Scaglietti, from northern Italy, found a higher incidence of congenital dislocations in the plain countries as compared to the mountainous areas. Of 3216 cases, Scaglietti mentions 84.7% were girls, 6.69% were hereditary, and 17.4% familial. Isigkeit assembled 12,764 cases from the literature - 85.7% girls, 15.2% hereditary, and 5% familial. Although the accuracy of these figures may be questioned, it seems significant that an appreciable per cent are hereditary of familial - about 20%. During Hitler's regime many studies were made to prove - not to ascertain - that all manner of congenital deformities were hereditary, the figures were large, but unreliable. The incidence of congenital hip dislocation in Italy and Germany was about 2 per thousand population. No figure is available for the United States, but I believe it would be much lower.

Other statistical reports in Scaglietti's series are of interest. From 1921 to 1931 there were 291 cases of subluxation. Comparing the figures for unilateral lesions and bilateral lesions, 45.9% were bilateral in dislocations and 10.65% only were bilateral in subluxations. Also of the 3,216 cases from 1899 to 1931, only 1.06% had associated congenital club feet. The presentation of the foetus at time of birth is no significance as an etiologic factor, since in 21,845 cases assembled by Pestalozza[3] 93.58% were cephalic, 4.28% breech, and 2.12% shoulder.

Etiology

In considering the etiology of congenital dislocation of the hip no proven theory exists. until recent years the emphasis has been placed on mechanical or traumatic factors, such as uterine deformities and constrictions and birth trauma. In an excellent article by Badgley,[4] failure of the normal embryologic rotation of the lower extremity or limb bud has been suggested as a factor. While this embyronic failure of internal rotation of the lower extremity or limb bud may occur, and while this may likewise be the explanation for the occurrence of congenital hips and equino-



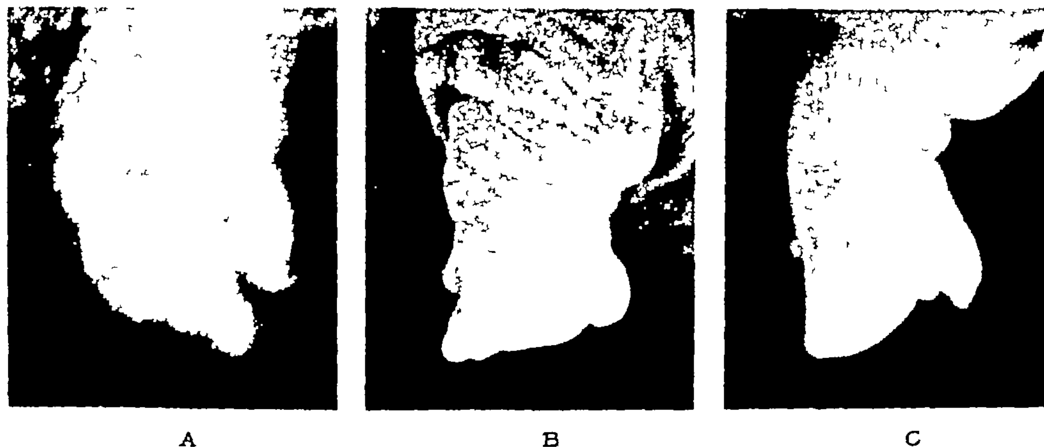
A B C

Fig 1. Congenital cleft palates of different etiology A. Hereditary cleft palate in a newborn mouse (After Steininger[7]) B Cleft palate of newborn rat whose mother was bred while fed diet deficient in riboflavin C Cleft palate of newborn rat whose mother was exposed to x-rays on the fifteenth day of gestation (Illustrations from J. Warkany)

valgus deformities, why is there failure of rotation?

It has been shown quite conclusively, especially by Warkany[5,6] that congenital malformations are only signs of prenatal pathology, due to one or more different causes. Congenital malformations in general, experimentally proven in rats and mice, may be hereditary,[7,8] owing to a riboflavin-deficient diet of the mother, or due to x-ray exposure of the mother rat on the 13th to 15th day after conception In the human, German measles (rubella) in the earlier months of pregnancy may cause various congenital malformations In cretinism, due to iodine deficiency, abnormalities of the skeletal system sometimes resemble some isolated congenital deformities

In Figs 1, 2, 3, and 4, through the courtesy of Dr Warkany, are shown cleared specimens and photographs of the newborn rats with congenital malformations (hereditary and phenocopies), and with lesions caused by riboflavin-deficient maternal diet or by x-ray exposure of the pregnant mothers on the 13th or 15th day In these illustrations can be noted the failure of rotation of the feet, various



A B C

Fig. 2. Three newborn hands with syndactylism of different etiology. A. Hereditary syndactylism in a mouse (After Bagg[8]) B. Syndactylism of newborn rat whose mother was bred while fed riboflavin-deficient diet C Syndactylism of newborn rat whose mother was exposed to x-rays on the 13th day of gestation (Illustrations from J Warkany.)



Fig. 3. Skeletal defects induced in newborn rats by exposure of the mother to roentgen rays during the period of gestation. A Exposure of mother on 12th day of gestation (Note large osseous defect in the skull). B. Exposure of mother on the 13th day of gestation (Note fusion of humerus and radius and shortening of ulna and fibula) C. Exposure of mother on the 15th day of gestation (Note angulation of ribs near vertebral junction) (Illustration from J. Warkany.)

mandibular, costal, radial and ulnar, tibial and tarsal phalangeal deformities. Close inspection of many of these congenitally deformed cleared specimens suggests the possible presence of some hip dislocations or abnormalities However, until tedious dissections of the hips have been done the incidence of the luxations of the hip cannot be said positively to exist in these specimens.

H. A. Harris[9] has stated that "The failure to maintain the hereditary form of the epiphysis, as a result of mucoid degeneration seen in achondroplasia and other specimens is probably the prime factor in congenital dislocations which are confined almost entirely to the slow growing ends of the bones at the elbow and hip." Following mucoid degeneration various types of deformities develop, seen as complete lack of growth to greater or lesser skeletal anomalies.

The obvious association of mucoid degeneration and experimentally produced congenital deformities must be considered as an etiologic explanation for human congenital deformities.

Pathology

Putti[10] has divided congenital hip dislocations into two types

1) embryonal or primary luxations, and
2) Foetal or birth luxations

From a pathologic and therapeutic aspect the second group, or birth luxations, should be subdivided into three groups a) subluxations, b) posterior dislocations, and c) anterior dislocations. The essential anatomic variations in the hip dysplasias, if we wish to so designate the entire group are

1) Acetabulum - from sloping or oblique

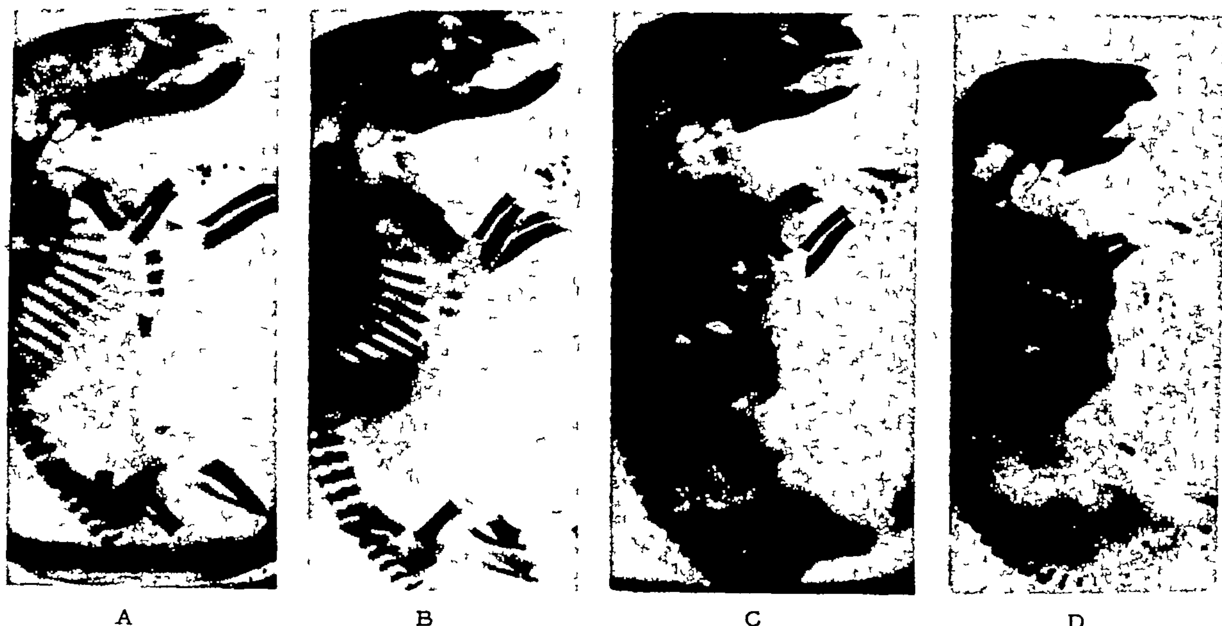


Fig 4 A. Cleared specimen of normal newborn rat B-D Clinical specimens of newborn rats whose mothers were bred while fed a riboflavin-deficient diet. Note short ribs in B, shortening of mandible, fusion of ribs, and absence of tibia in C, shortening of mandible, radius, and ulna, fusion of ribs, and absence of tibiae and fibulae in D (Illustrations from J Warkany.)



Fig 5 X-ray of newborn infant Congenital dislocation of both hips, embryonal or primary type. Note absence of capital femoral epiphyseal centers of ossification and external rotation of both legs.

superior margins with failure of development of lateral superior epiphysis, to complete absence with broad thick bone in the Y epiphyseal plate area A depression or defect may be noted laterally, posteriorly or anteriorly

2) Capsule - the capsule is elongated, having a narrow isthmus or constriction or, variably, may be redundant without a constriction It is frequently adherent to the ilium upward from the cotyloid ligament and may be adherent to the head of the femur superiorly, inferiorly, or circumferentially After the walking age the capsule hypertrophies superiorly.

3) Femur - early the femoral head, chiefly cartilaginous with delayed ossification, is spherical Later the head is misshapen due to failure of normal functional stimulation by the acetabulum.

Not infrequently osteochondritic or Legg's disease changes may be noted in the capital epiphysis These changes are noted prior to therapy almost as frequently as following it (See Fig. 11) It is generally assumed that the osteochondritic changes are due to circulatory changes, in luxated hips, either to the anomalous capsular attachments or to the trauma associated with abnormal function outside of



Fig. 6. X-ray of same case as Fig 5. Note congenital absence of radius and tarsal and metatarsal deformities

the acetabulum. An x-ray is shown with osteochondritic changes in the normal hip - without change in the luxated hip - indicating the lack of prima facie evidence



Fig 7 X-ray of two-year-old child (Case P. M.) Congenital lateral subluxation left hip. Note highly situated femur left, oblique roof of acetabulum. Shenton's obturator-coxo-femoral line is sketched in. On the right Shenton's line is continuous and normal On the left Shenton's line is broken and asymmetrical, diagnostic of a hip dislocation or subluxation



Fig 8. Same one-year-old case as in Fig 7, with legs externally rotated and abducted without anaesthesia. Left hip is now reduced.

A variable degree of anterior cervical torsion is almost always seen. On the x-ray this gives the impression of a coxa valga with a foreshortening of the neck, unless the leg be completely internally rotated In internal rotation a more exact appraisal of the degree of cervical torsion can be made. A subject for prolonged debates is the question Why is anterior cervical torsion present in both anterior and posterior dislocations? Badgley believes that all luxations are primarily anterior luxations and that later some become posterior luxations, probably embryologically or foetally.

Diagnosis[11]

The essential abnormalities will be outlined, but in order to make a diagnosis in the newborn or infant before the walking age has been reached, the single most important factor is to "think of it."

Diagnostic signs in the newborn or infant are

1) Asymmetrical creases in the adductor area of the thighs due to redundant soft tissue Fig. 13).

2) Shortening of the affected leg, determined by the flexed knee test (Fig. 14).

3) Widening of the perineum with prominent greater trochanters, especially noted in





Figs 9 and 10 (Case P. M , four-year-old child, same as in Figs 7 and 8.) X-rays, anterior-posterior of hips internally rotated and abducted and externally rotated Child was treated in left hip spica for six months Note lessened obliquity of acetabular roof, left, and normally developing hip joint, left, previously subluxated Hip joint was asymptomatic and there was no limp



Fig 11 (Case A R) X-ray of 2 1/2 year-old child with high posterior dislocation of the left hip Note left small capital epiphysis, extreme cervical torsion, deficient acetabular roof, and beginning formation of pseudoacetabulum in juxtaposition to head Note also early osteochondritis (Legg's disease) of normal hip

bilateral cases

4) Instability of the affected hip joint, noted on examination by a feeling of telescoping on the push and pull test, done with the hip extended, one hand grasping the thigh and the other hand stabilizing the pelvis

5) Limitation of extension of the hip joint in abduction for posterior dislocations, and increased extensibility of the hip joint in abduction for anterior dislocation

6) Relaxed hamstrings when the hip joint is abducted or abnormal extensibility of the knee joint in this position as compared to the normal leg

7) Palpable femoral head in the buttock or absence of the femoral head beneath the junction of Poupart's ligament and the femoral artery in posterior luxations In anterior luxations an abnormally prominent femoral head can usually be palpated anteriorly

8) X-ray examination Prior to ossification of the capital epiphysis the relations of the hip joint are more difficult to visualize, but asymmetry or a break in the hemispherical obturator-coxo-femoral line (Shenton's line)

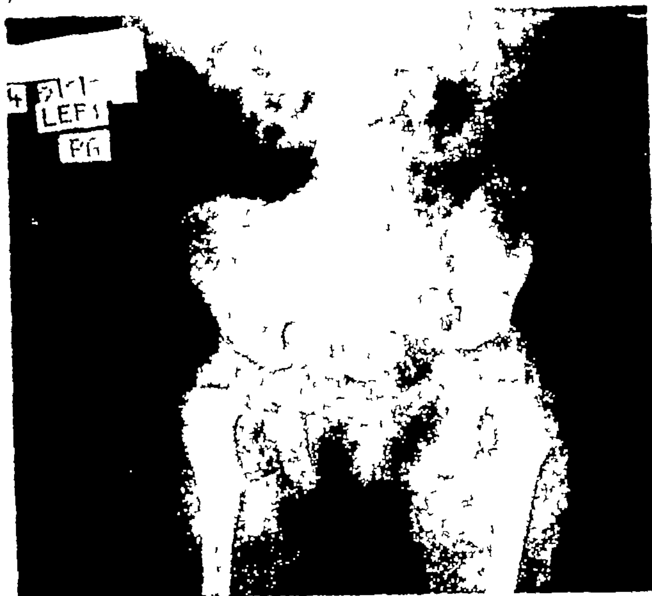


Fig. 12 (Case R. W.) X-ray of two-year-old girl with anterior dislocation of left hip. Note deficient acetabulum, cervical torsion, lateral misplacement of capital epiphysis, and early osteochondritic changes (Legg's disease) This child had also a congenital equinovarus deformity of the left foot.

is diagnostic (Fig. 7) The affected acetabulum has a more oblique roof or is more shallow, or both. The capital epiphysis, if ossified, is smaller There is an apparent coxa valga due to cervical torsion of the femur.

Diagnostic signs after the walking age

1) Increased lordosis or prominent buttocks is noted in posterior dislocations. Flattening of the lumbar spine and buttocks is noted in anterior dislocations

2) A rather typical gait consisting of a unilateral or bilateral sway due to relaxation or excessive length of the gluteus medius muscle or muscles, with a less obvious backward or gluteus maximus limp in posterior dislocations. The gait in anterior dislocations is less abnormal, but there is usually a moderate lateral waddle or gluteus medius limp.

3) Telescoping on the push and pull test of the affected hip or hips is readily noted excepting in older children when hypertrophy of the capsule may stabilize the hip in its malposition

4) The Trendelenburg test demonstrates the relaxation or apparent weakness of the gluteus medius muscle. When standing on the affected leg the patient can not lift the opposite side of the pelvis to or beyond the horizontal line, and

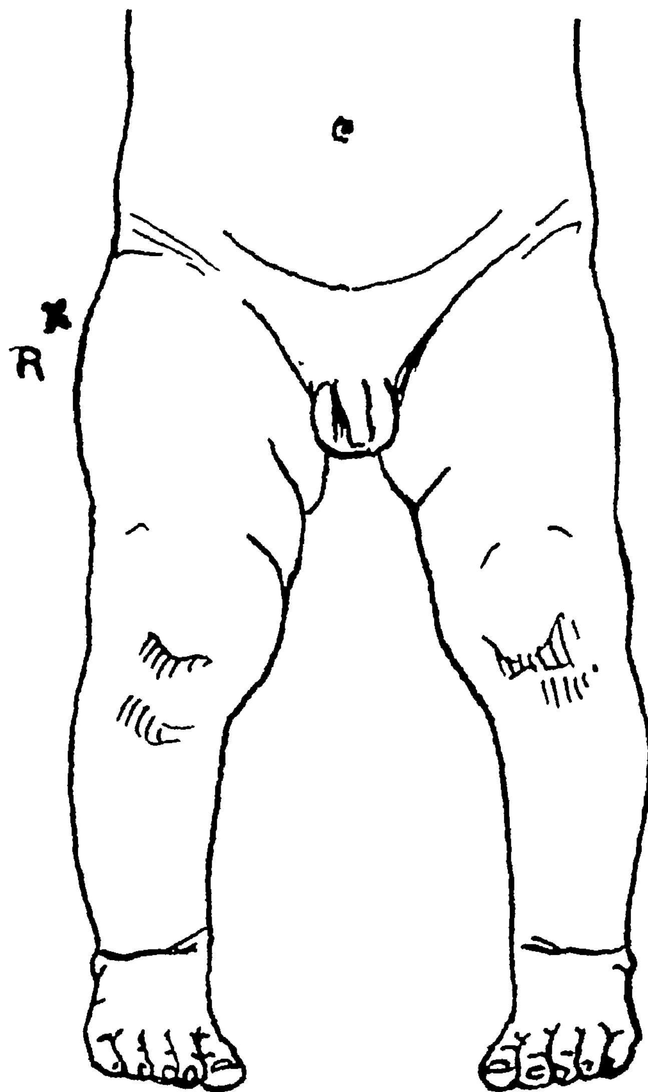


Fig. 13. Sketch of a newborn infant with legs placed parallel. Note on right side fullness in greater trochanter area and two skin thigh creases contrasted with a single shallower thigh crease on the left These findings are always suggestive of a right congenital hip dislocation or subluxation

must shift his or her center of gravity laterally beyond the longitudinal axis line of the affected hip

5) X-ray examination will demonstrate the abnormal relation of the femoral head and the acetabulum and the characteristic acetabular and femoral head and neck abnormalities.

Differential Diagnosis

The following lesions or conditions must be considered for the stated reasons

1) Congenital coxa vara, also osseous short-

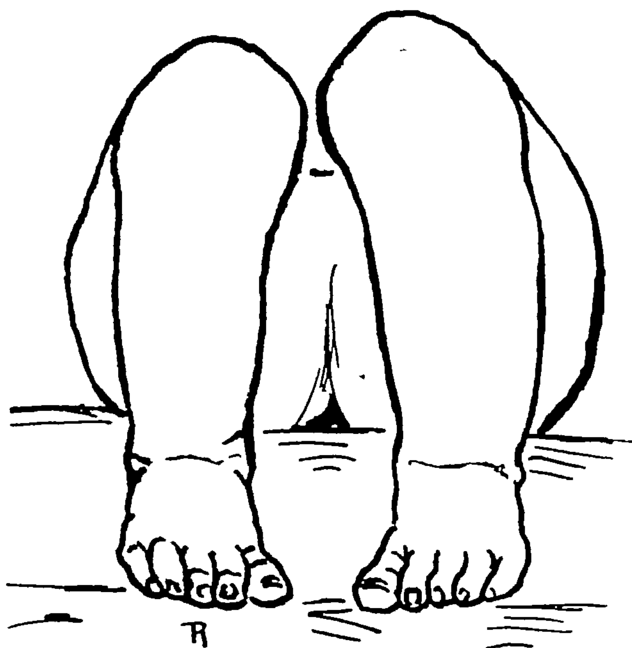


Fig 14 Sketch of a newborn child with a congenital dislocation of the right hip Difference in leg length is difficult to determine at this age with tape line With the child on its back, the knees moderately flexed and the feet held parallel and together, shortening is demonstrated by the difference in knee levels, the right being lower, indicating a shortening of the right leg

ening, a slight lateral waddling gait, but no true instability.

2) Absorption of the femoral head from infection, similar gait, telescoping, positive Trendelenburg test, highly placed greater trochanter, but femoral head not palpable X-rays are diagnostic

3) Poliomyelitis with residual paralysis of gluteus medius, or gluteus maximus, or both muscles Only similarity on examination is the limp unless there is an associated pathologic dislocation associated with the muscle paresis, in which case, as in pathologic dislocation following an acute infectious process, x-ray examination will fail to show the typical acetabular and femoral head and neck abnormalities seen in congenital dislocations

4) Neurotrophic lesions of the hip joint, though rarely seen in children or young adults, may present many of the clinical signs of congenital dislocation, at least those characterized by instability of the hip joint The neurologic and x-ray findings will facilitate the differential diagnosis in these cases

5) Ununited fracture of the neck of the femur (this may occur in children as well as in adults) has all of the signs associated with instability of the hip joint, telescoping, lateral list gait, and Trendelenburg sign, but the history and x-ray findings are diagnostic

REFERENCES

1 Scaglietti, O in Chir d Org di Movimenti, 17 225-243 (Aug , 1932)
2 Isigkeit, E in Arch f Orthop 26 659-718 (Oct , 1928)
3 Pestalozza, quoted by Scaglietti
4 Badgley, C E in J Bone & Joint Surg 25 503-523 (July, 1943)
5 Warkany, J Some Factors in the Etiology of Congenital Malformations Amer J Mental Deficiency 50 231-241 (Oct , 1945)
6 Warkany, J , et al J Bone & Joint Surg 25 261-270 (April, 1943)
7 Steininger, F Neue Beobachtungen an der erblichen Hasenscharte der Maus Ztschr f mensch Vererb u 23 425 (1939)
8 Bagg, H J Hereditary Abnormalities of the Limbs, Their Origin and Transmission Amer. J Anat 43 167 (March, 1929).
9 Harris, H A Bone Growth in Health and Disease Oxford University Press, 1933
10 Putti, V in Chir d Organ di Movimenti 17 209-217 (August, 1932)
11 Freiberg, J A Early Diagnosis and Treatment of Congenital Dislocation of the Hip J A M A 102 89-92 (Jan 13, 1934)

EARLY MANAGEMENT OF CONGENITAL DISLOCATION OF THE HIP

Classification of Congenital Dislocation of the Hip with the Treatment of Each Type from Infancy to Age of Eight Years

H. R. McCarroll, M D

Congenital dislocation of the hip still represents one of the most interesting problems encountered in crippled children's work. Generalities are difficult in the discussion of a treatment of this disorder and every case must be considered as an individual problem, which frequently taxes the ingenuity of the surgeon. In a discussion of this type certain generalities must be made, however, in an attempt to formulate a rationale of treatment, but it should be remembered that in any given case unusual difficulties may be encountered necessitating a change in the plan of therapy according to the situation presented.

The period between birth and the age of eight years represents the period during which treatment in the child with congenital dislocation of the hip is able to restore the highest percentage to normal anatomical relationships and insure normal subsequent development and perfectly normal function for the hip joint. This aim from the standpoint of treatment is not fulfilled in all cases, but the percentage of satisfactory results during this age group is quite high and much greater than that which is possible beyond the age of eight. In this age group every attempt should be made to restore the hip to its normal anatomical position. If this fails, any of the less desirable reconstructive procedures applicable in older children can still be used in these patients after they are beyond the age group of possible normal anatomical restoration.

Management of Congenital Dislocation of the Hip from Birth to Walking Age

The diagnosis of congenital dislocation of the hip in infancy is possible but seldom made. The first evidence of difficulty in these children is usually encountered after walking has begun Accurate diagnosis is possible, however in infancy, and even at the time of birth, if the child is carefully examined with this possibility in mind There are four points which may be helpful in establishing a diagnosis at this age 1) telescoping of hip, the only reliable sign. 2) x-ray change, 3) extra skin crease in thigh, and 4) shortening. The only accurate sign is demonstrable telescoping of the femur or hip as demonstrated in Fig 1. X-ray chang-

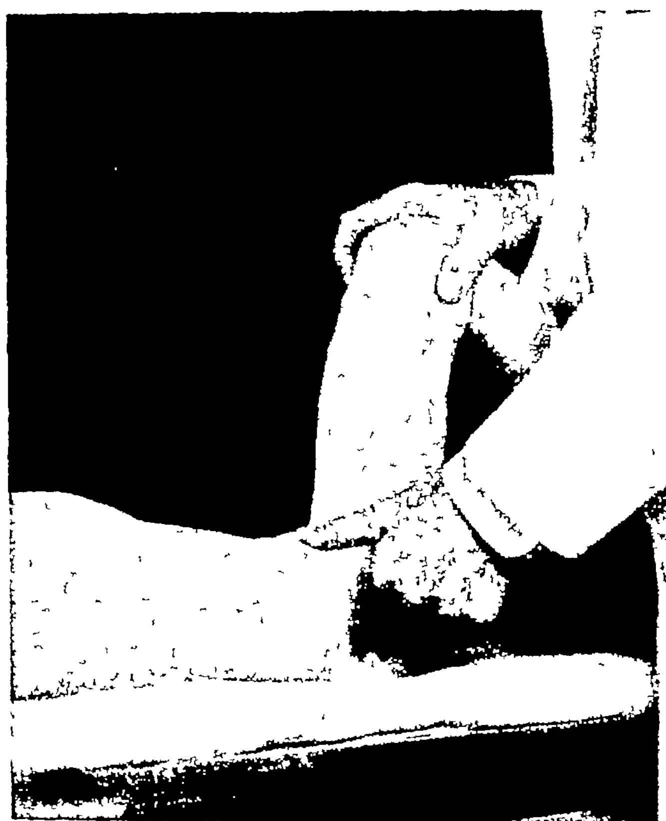


Fig 1 Photograph showing method of demonstrating telescoping of the hip in the presence of a dislocation Pistonlike action of the head of the femur along the pelvis can be shown in this way.

es in infancy are usually not accurate because the upward displacement of the femoral head has not yet occurred to any great extent and because the center of ossification for the femoral head has not appeared and the bony components of the hip are sometimes difficult to outline. The extra skin crease in the thigh also results from upward displacement and is therefore frequently not present. An extra skin

crease can at times be seen in a normal extremity, and the sign is, therefore, not entirely reliable Shortening of the involved extremity may be present, but here again it is usually absent at this age since upward displacement of the femoral head has not yet occurred to any great extent This sign is, of course, worthless in a bilateral involvement

Attempting to differentiate the type of dislocation in a patient of this age is of little or no importance This is usually impossible and treatment is the same for all patients, regardless of the type of dislocation In infancy, treatment should be instituted at once Since appreciable upward displacement of the femoral head has not yet occurred, forceful manipulation and traction of the hip is as a rule not necessary Since the greatest malposition consists of simple lateral displacement of the femoral head, the hip can frequently be reduced by simple abduction and internal rotation of the extremity with a little manual traction This should be attempted and the hip should be immobilized, maintaining this position A frog position is not used and is not necessary, in fact it is not anatomically correct as far as the deformity with which we are dealing is concerned Fixation of these hips afterwards is maintained by means of plaster casts immobilizing them in the position of abduction and internal rotation In the beginning this should consist of a hip spica extending from above the lower rib margin to the toes on the involved extremity and to the knee on the opposite extremity (Fig 2) The plaster should be changed at relatively frequent intervals, on an average of 4 to 6 weeks because of the danger of irritation of the skin and the difficulty of maintaining cleanliness in a child of this age It is impossible to state with any degree of accuracy the length of immobilization necessary for a given child The period of fixation should be maintained for a minimum of 6 months unless one can be certain that a normal position has been assumed and complete stability of the hip can be demonstrated If fixation is maintained beyond this time, long leg plaster casts and crossbars, as shown in Fig 3, may then be substituted for the plaster spica This will allow motion in the hips, maintaining at the same time the fixed position of abduction and internal rotation It also facilitates the care of the child a great deal



Fig. 2 Bilateral hip spica showing method of postreductive fixation

The above discussion is a purely theoretical one as far as I am concerned I have never seen congenital dislocation of the hip in an infant I have watched for it closely in the patients that I have had an opportunity to examine in infancy, but the youngest patient I have seen with this condition is 11 months of age Fig. 4A shows the x-ray of a newborn baby in whom a unilateral dislocation of the hip was suspected In spite of the suggestive appearance of the x-ray, no telescoping of the hip could be demonstrated and it was, therefore, concluded that the hip was not dislocated This was substantiated by x-ray at the age of 18 months (Fig 4B), which revealed normal hip joint structure, identical on the two sides, even though no treatment was used



Fig. 3. Bilateral long leg plaster casts with the hips held in abduction and internal rotation by means of crossbars This is used as a means of postreductive fixation in certain instances.



Fig. 4A. X-ray of pelvis in newborn infant in which congenital dislocation of the hip was suspected on the right Even though the right hip seemed to be displaced laterally, telescoping could not be demonstrated and possibility of dislocation was in this way excluded.



Fig 4B. X-ray of pelvis of same infant 18 months later Both hips are normal in appearance. No treatment was used

Diagnosis and Classification of Congenital Dislocation of the Hip from Walking Age until 8 Years of Age

During the first year of life active use of the extremity by the patient will usually result in definite upward displacement of the femoral head due to the muscle pull and, at times, actual contracture of the muscles about the hip joint The average child begins to walk about the age of 1 year, and after weight bearing on the extremity is once begun, the upward displacement of the femoral head increases as a result of this force. It is at this age that the diagnosis of congenital dislocation of the hip is usually first made. The usual complaint of the mother at the first examination is that a limp was noticed as soon as the child began to walk Diagnosis of the condition is relatively simple at this age since in addition to the limp and demonstrable telescoping, the child usually shows some shortening, a positive Trendelenburg, and a trochanter which rests above Nelaton's line

The exact clinical picture which the patient presents at this time depends upon the type of dislocation which is present There are three clinical types of congenital dislocation of the hip, each of which exists as a distinct clinical entity. The classification of these various types is not only of importance from an aca-

demic standpoint but, from the standpoint of treatment which must be considered These three clinical types are shown in Table I together with their relative frequency The total of 111 cases represents the number of cases treated at the St Louis Unit, Shriners' Hospital for Crippled Children from 1935 to 1945 inclusive.

Table III

Clinical Features of Primary Anterior Dislocation of the Hip
1 Flat Buttock
2 Flat Lumbar Spine
3 Prominence in Femoral Triangle
4 Positive Trendelenburg
5 Trochanter May Not Be above Nelaton's Line
6 Less Shortening
7 Less Limp

The clinical features in the different types of dislocation depend on the position of the femoral head in relation to the acetabulum and the line of weight bearing The relationship of the trochanter to Nelaton's line is of no significance in determining the clinical characteristics presented by these patients The clinical features of the posterior type are shown in Table II. This represents the classical textbook picture of congenital dislocation

Table II

Clinical Features of Primary Posterior Congenital Dislocation of the Hip
1 Prominent Buttock
2 Exaggerated Lumbar Lordosis
3 Flat Femoral Fossa
4 Trochanter above Nelaton's Line
5 Positive Trendelenburg
6 Shortening
7 Limp on Walking

of the hip and is by far the most common type encountered. In this type, the buttock is quite prominent due to the fact that it is bulged by the mass of trochanter high in a posterior position (Fig 5, 6A, 9A, 12A, and 13B) Since the femoral head is posterior to the line of weight bearing, the pelvis is rotated forward with the head of the femur serving as the pivot point With the pelvis rolled forward, obvious-



Fig. 5. Photograph of patient with unilateral posterior congenital dislocation of hip Note prominent buttock and increased lumbar lordosis

ly the entire lower spine is tilted forward In order to maintain an erect position the upper part of the spine is pulled posteriorly, resulting in an exaggerated lumbar lordosis. The flattening of the femoral fossa anteriorly is due to the fact that the entire mass of the hip joint is dislocated posteriorly and there is a subsequent depression at this point The other clinical features, positive Trendelenburg, a trochanter which rests above Nelaton's line, shortening in a unilateral involvement, and a limp on walking, are not important in differentiating one type of dislocation from another. These features, although more marked in the posterior type, are usually present in varying degrees in every type.

The clinical features of the primary anterior type of dislocation of the hip are shown in Table III The picture which this patient presents is to a large degree the exact opposite of that encountered in the primary poste-

Table I

Three Clinical Types of Congenital Dislocation of the Hip	Total Number 1935 - 1945
Primary Posterior Dislocation (Classical picture of dislocated hip)	68
Primary Anterior Dislocation	25
Simple Upward Subluxation	18
Total	111

rior type of dislocation. The Trendelenburg sign, the relationship of the trochanter to Nelaton's line, the degree of shortening, and the limp which the child shows are usually not as marked in this type as they are in the primary posterior dislocation. This is due to the fact that the femoral head, instead of being displaced posteriorly, is displaced anterior to the acetabulum and rests in the fossa just beneath the anterior superior iliac spine. As a result of this, the femoral head is held more securely in position and the degree of shortening is appreciably diminished. Since the femoral head is displaced into a true anterior position, the pivot point is anterior to the line of weight bearing and the pelvis is rolled backward, and since the pelvis is rolled backward by this anterior thrust, the lumbar spine is flattened (Figs. 7, 10A, 12B and 13A). The lumbar lordosis is never exaggerated in the primary anterior type of dislocation, is frequently less than normal, and many patients will present an entirely flat lumbar spine. With the femoral head displaced anteriorly, the buttock on this side is flatter than normal. The femoral fossa anteriorly instead of being flat is more prominent than that on the normal

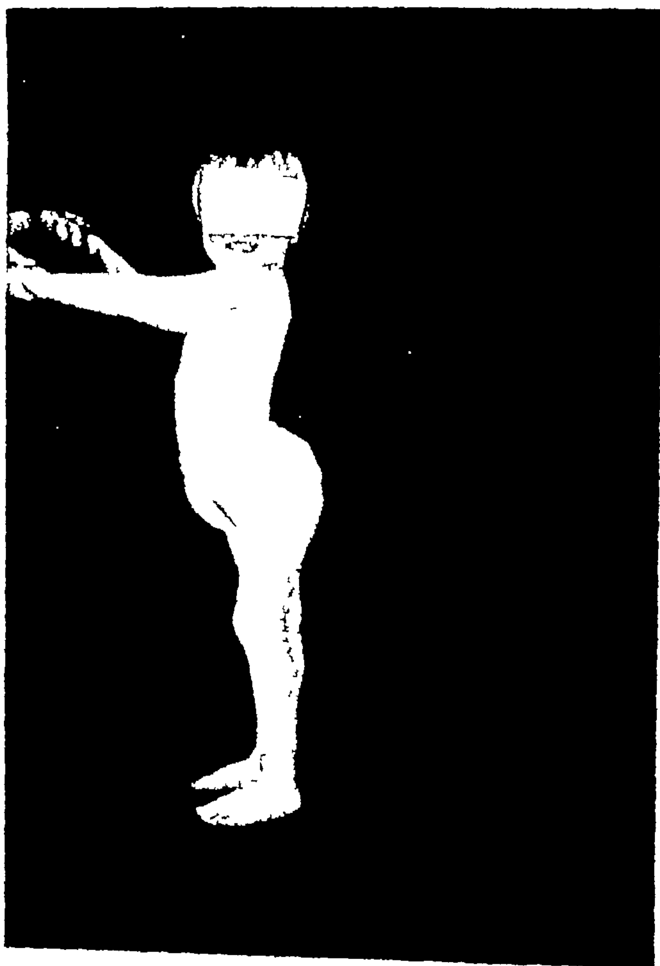


Fig. 6A. Photograph of patient with bilateral posterior congenital dislocation of hip Note prominent buttock and exaggerated lumbar lordosis.



Fig. 6B. Photograph of same patient following treatment in which closed reduction was used for each hip. Note the normal appearance of the buttocks and the normal degree of lumbar curve as compared with Fig. 6A.

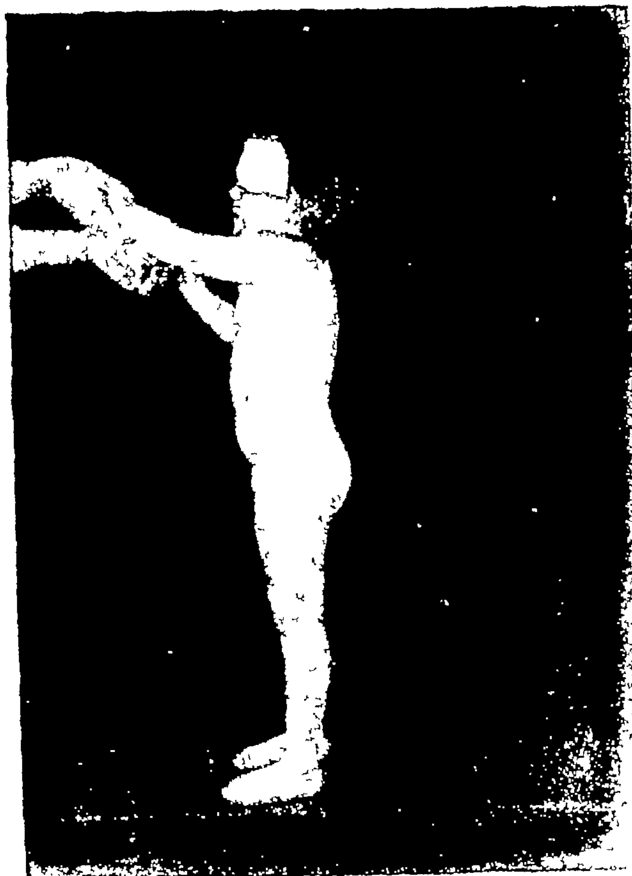


Fig. 7. Photograph of patient with unilateral primary anterior congenital dislocation of hip Note the lack of prominence in the buttock and the relatively flat lumbar spine even though a dislocation is present.

side since the femoral head and the entire mass of the hip joint is displaced anteriorly to produce a bulge at this point. In heavy children this sign may be difficult to observe.

The clinical features of the simple upward subluxation are shown in Table IV This hip is not displaced anteriorly or posteriorly, but simply displaced upward to a varying degree because of an inadequate acetabular roof. In this type of dislocation there is a primary developmental defect in the superior rim of the acetabulum, and the ledge beneath which the head of the femur rests is insufficient to maintain stability and a fixed position It may not even be a true dislocation (Fig. 11B) The femoral head may still rest in the acetabulum and tend to wander upward only after years of weight bearing This position accounts for the clinical characteristics of this type of involvement Since the hip is not displaced anteri or posteriorly, the buttock may be norma degree of flattening as compared with the n

Table IV

Clinical Features of Simple Upward Subluxatio
1 Normal Buttock
2. Normal Lumbar Curve
3. Questionable Trendelenburg
4 Trochanter in Normal Position.
5 Little if any Shortening at this Age
6. Usually no Limp at this Age.

mal side (Fig. 11A) Since there is no char in the line of weight bearing, the lumbar lo dosis usually remains normal in degree. the earlier years, little or no shortening m be present and as a result of this the child m exhibit very little, if any, limp Since there little or no shortening, the trochanter may r main in normal position in relation to Nelaton line and the Trendelenburg sign may even questionable

This classification into three types of di location, as stated, is based entirely on t clinical picture which these patients present The x-ray is of no value in classifying these hips except in the case of the simple upwar subluxation. Attempts have been made to obtain lateral x-rays, as shown in Fig 8, bu this has proven to be a difficult task, and accurate interpretation of the x-ray is not always possible Stereoscopic views have also bee used, but here again the exact position of the femur in relation to the acetabulum cannot always be determined From a clinical standpoint, however, the classification of the hip is usually possible and should be attempted at the time of the first examination in order to have clearly in mind the various steps which may be necessary in the subsequent treatment

Treatment of Congenital Dislocation of the Hip from Walking Age to 8 Years of Age

In general, the treatment of congenital dislocation of the hip for this age group may be stated simply as reduction of the hip into the acetabulum with fixation in this position long enough to insure this as a permanent position for the femoral head. Beyond this point generalities are difficult or impossible in the treat-

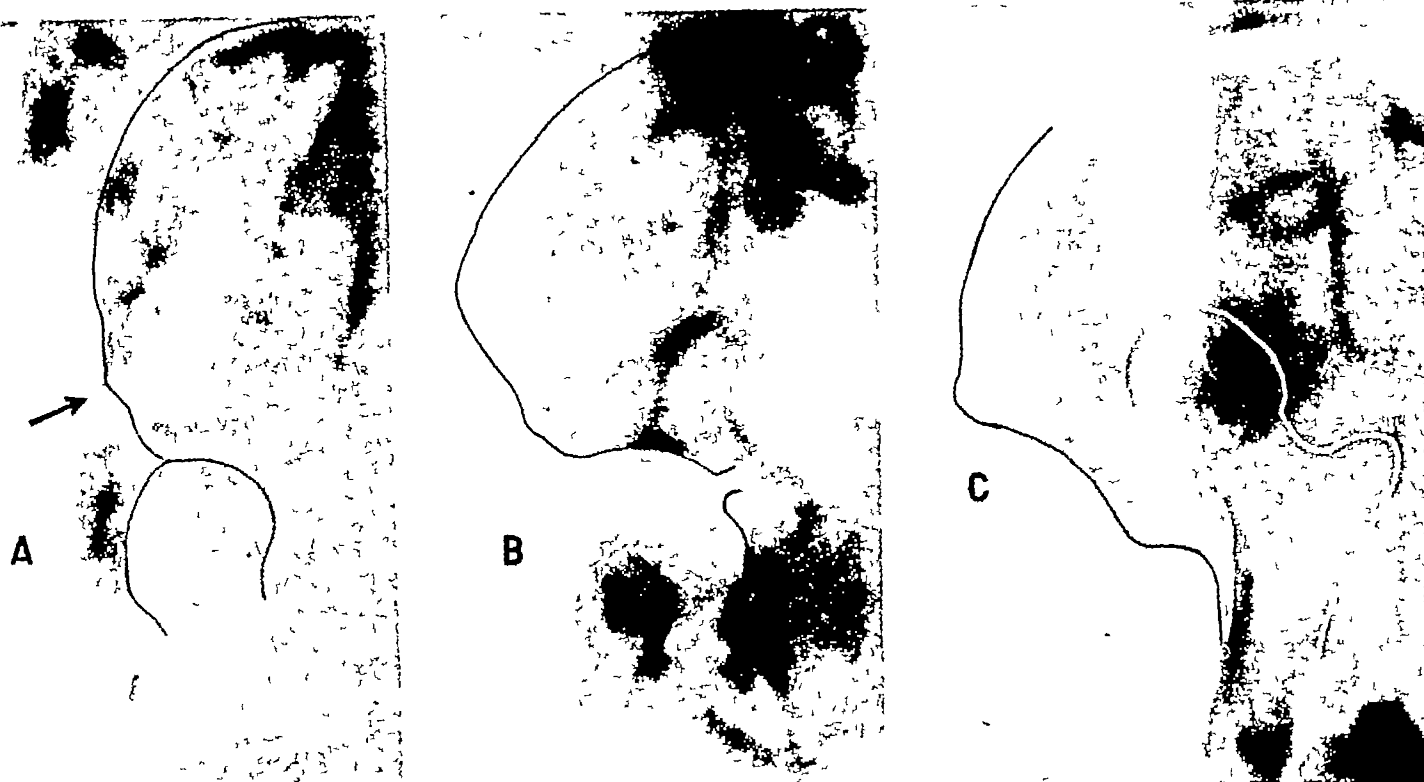


Fig. 8. Lateral roentgenograms of hips, showing relationship of femoral head to anterior superior spine. A. Primary anterior dislocation of hip. The femoral head is resting below the anterior superior spine, which is marked by the arrow The Pelvis is rolled backward as the result of the anterior thrust in the line of weight-bearing B. Lateral view of normal hip in same patient C. Lateral view of posterior dislocation of hip The femoral head in this instance is resting against the wing of the ilium far posterior to the anterior superior spine. The pelvis is rolled forward as the result of the posterior thrust in the line of weight-bearing. (From J. Bone & Joint Surg. 21 648-664, 1939.)

ment of this disorder. The treatment may be vastly different in the three different types of dislocation and for that reason additional discussion of the treatment of each will be given separately.

One of the oldest forms of treatment for congenital dislocation of the hip has been manipulation of the hip under an anaesthetic and fixation in plaster-of-paris spica for many months. In reviewing the literature on this form of treatment, one is impressed with the frequency with which stress is placed on gentleness in the manipulation of these hips in order to avoid trauma. Any experience with this form of treatment soon convinces one, however, that gentle manipulation cannot always be adhered to if reduction of the hip is to be brought about It is not unusual to have a great deal of hemorrhage, ecchymosis, and swelling appear subsequent to such a manipulation as an indication of soft tissue damage and tear. Great force must often be exerted in traction on these extremities and in ironing out the contracted muscles about the hip joint in attempting to force the hip into its normal anatomical relationship. If soft-tissue damage results in this way, it is quite possible that considerable damage to the bony components of the hip joint may also follow such therapy. The articular cartilage and the unossified capital epiphysis are soft and friable structures in a young child, and damage in a manipulation of this type may easily occur. Such a reduction results in tremendous intra-articular pressure which exists over a period of days This is due to the fact that the contract-



Fig 9A. Photograph of untreated bilateral posterior congenital dislocation of hip in an adult Prominent buttock and exaggerated lumbar lordosis are apparent

ed muscles, forced very rapidly into a stretched position, are still under great tension and tend to contract to their original length. This in turn exerts great force of the femoral head against the acetabulum and may even be sufficient to cause some vascular change in the immature capital epiphysis. The trauma of forceful manipulation and the increased intra-articular pressure which follows may easily account for the marked secondary degenerative changes which are often encountered in these hips in later years These changes are comparable to a traumatic arthritis or in some instances to an old aseptic necrosis of the femoral head. Treatment of the hip by forceful

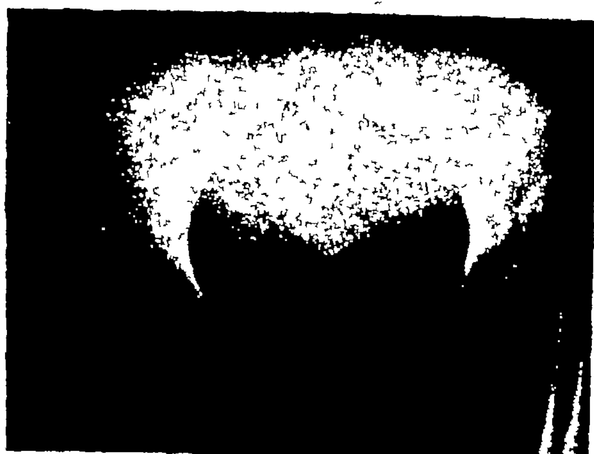


Fig 9B X-ray of patient shown in Fig 9A

manipulation has not, therefore, given entirely satisfactory results

It was because of this fact that Crego in 1931 first instituted skeletal traction as a preliminary procedure in the treatment of young patients with congenital dislocation of the hip Skeletal traction had been used in older patients preliminary to arthrodesis or other reconstructive procedures, and Crego reasoned that, if this was possible in older children, the same procedure could be followed in younger children and reduce the incidence of trauma Preliminary skeletal traction has, therefore, been used as a routine procedure at the St Louis Unit, Shriners' Hospital for Crippled Children, since 1931 and has appreciably reduced the incidence of secondary degenerative changes encountered in these hips This procedure has facilitated the management of these patients and has made it possible to bring about reduction of the hip without force and often without any manipulation whatever Spontaneous reduction of a dislocated hip in skeletal traction is not an infrequent occurrence An anaesthetic is seldom needed at the time the traction is discontinued and plaster fixation is instituted, except in the very young children where adequate cooperation is impossible In the beginning, skin traction was tried but this was found to be inadequate In a young child it is not uncommon to require 15 or 20 pounds of weight for adequate reduction of the hip In some children traction equivalent to the body weight may be required Skin traction in a young baby will not tolerate more than 4 or 5 pounds and cannot be continued



Fig. 10A. Adult patient with untreated unilateral primary anterior congenital dislocation of the hip. Note the extremely flat buttock on the involved side as compared with the bulge of the normal buttock on the opposite side The femoral head and anterior superior spine are outlined Note the relatively flat lumbar spine.



Fig 10B X-ray of patient shown in Fig 10A.

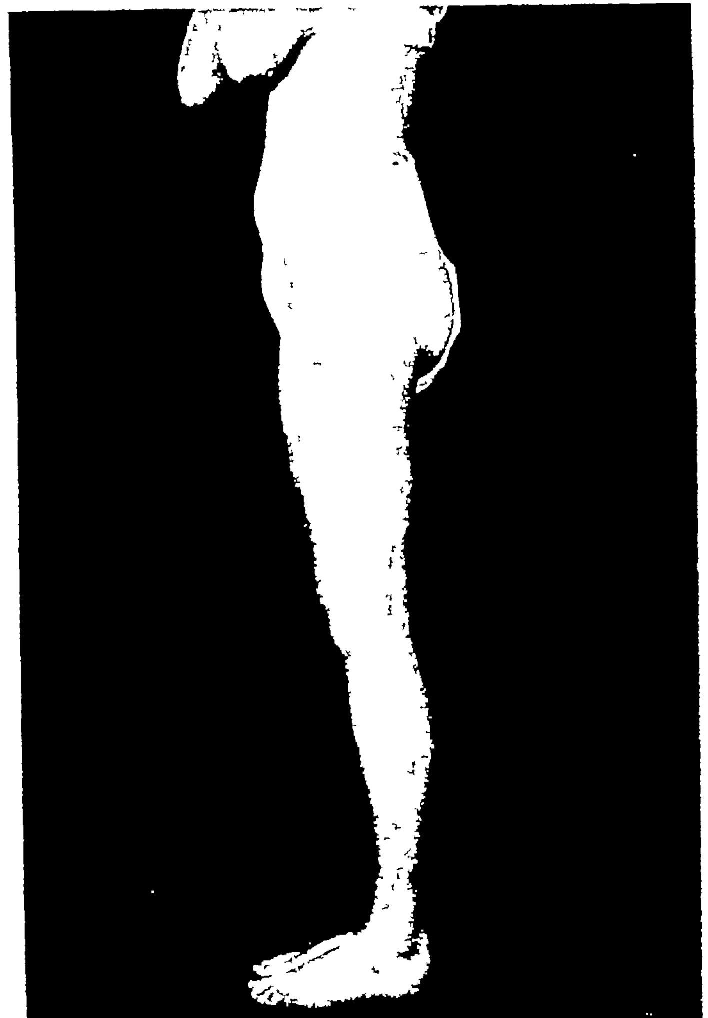


Fig. 11A. Adult patient with untreated unilateral simple upward subluxation. Note the relatively normal buttock and the normal lumbar curve.

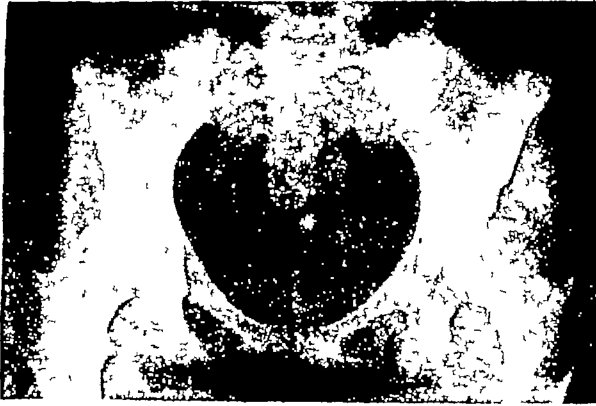


Fig 11B X-ray of patient shown in Fig 11A Simple upward subluxation with the femoral head riding approximately halfway out of the acetabulum





A B

Fig 12A Lateral view of child with bilateral posterior congenital dislocation of the hip Note the marked exaggeration in her lumbar lordosis
Fig 12B Lateral view of child with unilateral anterior congenital dislocation This child shows a perfectly flat lumbar spine without even the normal amount of lumbar curve usually seen



A B

Fig 13A Unilateral primary anterior congenital dislocation of the hip Note flat buttock on involved side and the relatively flat lumbar spine
Fig 13B Bilateral posterior congenital dislocation of the hip Note extremely prominent buttock and exaggerated lumbar lordosis

over a long period It was found essential, therefore, to use skeletal traction as a routine procedure This is accomplished by drilling a wire through the lower end of the femur above the epiphyseal line, the addition of a traction bow, and the application of sufficient pull to bring the femoral head well below the superior rim of the acetabulum Body weight is used as counter traction and is obtained by elevation of the foot of the bed (Figs 14 and 15) A reduced position may be obtained in some instances within 10 or 12 days The traction, however, is maintained for a minimum of 3 weeks in order that the soft tissue structures about the hip joint may be adequately relaxed In this way there is no increased intraarticular pressure from the pull of these soft-tissue structures following reduction

In speaking of generalities, two other points may be covered as being common to all types in most instances The first of these is the position of postreductive fixation In past years

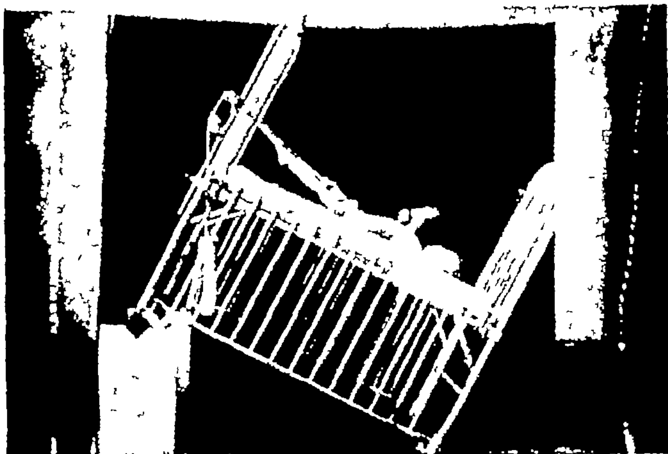


Fig. 14 Photograph showing method of skeletal traction in an infant. Body weight serves as a counter traction on elevation of the foot of the bed

the frog position has been the position most commonly used. In this the hip is flexed, abducted, and externally rotated in order to lift the femoral head forward into the acetabulum One is impressed, however, in hips adequately reduced by preliminary skeletal traction with the fact that the position of greatest stability is one of simple abduction and internal rotation. As a result of this finding, the frog position has long been discontinued. These hips, regardless of the type, are immobilized in a bilateral hip spica maintaining the involved extremity in a position of abduction and internal rotation. This position is shown in Fig. 2.

The second generality deals with the correction of the associated femoral torsion (Fig. 16E). If a hip is maintained in the position of internal rotation for 2 to 3 months, which represents the usual period of postreductive fixation, and then liberated for active use, the child will invariably externally rotate the foot to the midposition rather than maintain the position of fixed internal rotation In doing so the femoral head is rotated forward and often redislocates through the anterior rim of the acetabulum as a result of this rotating force. There are claims in the literature that this internal torsion will correct itself spontaneously. This may be true if the position is maintained over a long period of time. It will not correct itself, however, in the usual period of fixation extending over 2 or 3 months. It seems reasonable to assume, therefore, that this torsion should be corrected in order to maintain the position of internal rotation, which represents the position of stability, and thus prevent subsequent redislocation of the hip.

Treatment of Primary Posterior Congenital Dislocation of the Hip

The routine in use for these hips at present is as follows· A wire is inserted through the lower portion of the femur, and skeletal traction is instituted. The position of the femoral head is observed by frequent x-rays and the amount of weight required is determined by the degree and speed with which the femoral head is brought down to a point opposite the acetabulum. The amount of weight required to accomplish this is added. Traction is maintained over a period of 3 weeks in all cases. In those cases which are resistant to reduction and require essentially 3 weeks to bring the femoral head into satisfactory position, the traction is maintained for 4 weeks or longer in order to insure adequate relaxation of the soft tissue structures. Following this period of traction, the wire is removed and one finds

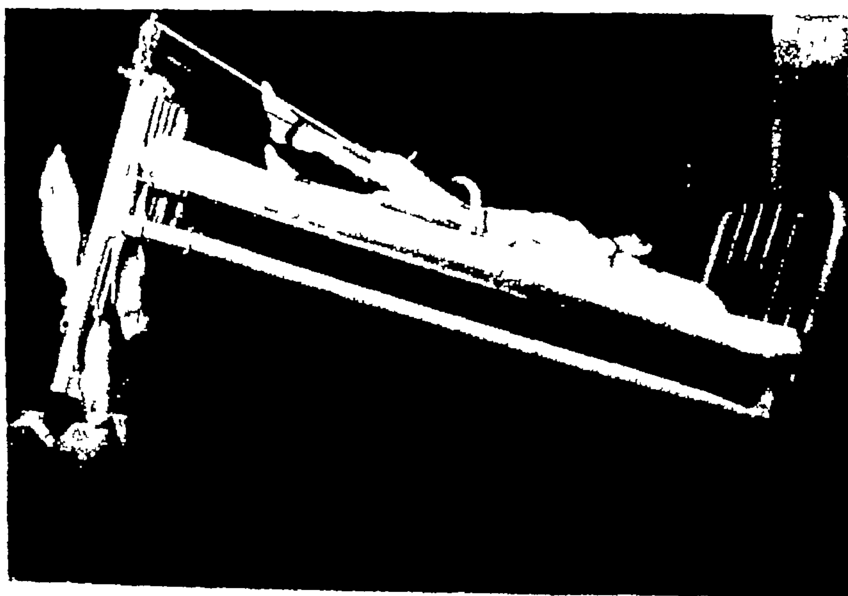


Fig. 15. Photograph showing method of skeletal traction in an older patient.

that the hip can be gently moved back and forth without force, easily slipping the head of the femur in and out of the acetabulum. No anaesthetic is required for this procedure unless the child is extremely young and adequate co-operation is impossible Invariably the position of stability is found to be one of abduction and internal rotation. The hip is maintained in this position and a bilateral hip spica is applied (Fig 2)

By x-ray observation and by the degree of stability demonstrated at the time of reduction, the degree of adequacy and the depth of the acetabulum can usually be determined If an adequate acetabulum is present, surgery in the hip itself can usually be avoided The only exception to this rule lies in that occasional case where the acetabular fossa may be completely filled with a mass of fibrous tissue In this event open reduction is necessary and the mass of fibrous tissue must be removed from the acetabulum before the femoral head can be adequately seated The presence of a mass of fibrous tissue in the acetabulum cannot, of course, be accurately diagnosed prior to operation In the presence of a roentgenologically adequate acetabulum it can be suspected, however, if at the time of reduction the femoral head cannot be felt to slip over a definite rim and it cannot be deeply seated in the acetabular fossa. This represents one indication for open reduction in these patients, but unfortunately this type of involvement is relatively rare. If open reduction is not indicated, the position of abduction and internal rotation is maintained for 2 months At this time the plaster is removed and a supracondylar osteotomy (Fig 16E) is performed in order to correct the torsion which is invariably present This is accomplished by maintaining the extremity in a position of internal rotation after the plaster is removed and until the extremity is prepared and draped for operation. The degree of internal rotation frequently approximates 90° With the extremity held in this position a wire is drilled through the femur three inches above the lower femoral epiphyseal line keeping this wire parallel to the operating table The horizontal position of this wire, therefore, marks the desired position of internal rotation to be maintained for the hip A bow is applied to this wire in order to maintain the position of internal rotation A transverse supracondylar osteotomy is then performed through a lateral incision two inches in length just below the point of the wire The hip is maintained in a position of internal rotation by grasping the bow attached to the wire and maintaining the wire parallel with the operating table The lower extremity is then grasped at the knee and externally rotated, the point of rotation taking place at the site of the osteotomy It is externally rotated until the knee, ankle, and foot are in proper alignment with the anterior superior iliac spine The wound is then closed and a unilateral hip spica is applied, maintaining this position and incorporating the wire and bow in the plaster cast (Fig 16E). Immobilization in this plaster cast is again maintained for 2 months At the end of this time the wire is removed and the patient is allowed active motion and use of the extremity as rapidly as desired Usually no difficulty is encountered in restoring these hips to a satisfactory range of motion and the average child will accomplish this without physical therapy within 2 or 3 months (Figs. 16, 17, 18, and 19)



Fig 16A X-ray of unilateral primary posterior congenital dislocation of hip In this x-ray the knee on the involved side is pointing directly forward Note almost complete absence of angle of femoral neck This is due to the presence of femoral torsion

The most common indication for open reduction in the posterior type of dislocation is the presence of an acetabulum which is inadequate to maintain the position of the femoral head (Figs. 20C, 21A, 22A, and 23A) This must be determined from the appearance of the x-ray and the feel of the hip at the time of reduction If the acetabulum is known to be in-



Fig 16B. X-ray taken at the same time with the knee on the involved side internally rotated 90°. Normal femoral neck and normal angle of the femoral neck is in this way demonstrated. Compare with Fig 16A The difference in the angle of the neck is represented by the amount of femoral torsion which is present

adequate, there is no reason to postpone open reduction If, however, there is some question about the status of the acetabulum, closed reduction followed by derotational osteotomy should be performed first In the event the acetabulum still proves to be inadequate and the hip redislocates, open reduction and acetabular reconstruction, shelf operation, can then be carried out. The procedure of acetabular reconstruction is demonstrated in Figs. 20A to 20D This consists first of adequate exposure of the hip joint and acetabulum. If there is any question about the position of the femoral head in the acetabulum, the joint capsule should be opened and the hip joint inspected. Once the position of the femoral head is adequately proven, the joint capsule is again closed and the remainder of the procedure is done entirely extracapsularly. By means of an osteotome a rim of bone is raised from the ilium just above and parallel to the superior rim of the acetabulum It is levered into a horizontal position and blocked in this position by packing into the defect just above it chips of cancellous bone removed from the wing of the ilium. Here again the extremity is reduced and maintained in a position of abduction and internal rotation. The wound is closed and a bilateral hip spica applied, maintaining this position. Following open reduction, plaster fixation is continued for 3 months instead of



Fig 16C. The same hip with skeletal traction applied showing the femoral head pulled well below the superior rim of the acetabulum.

2 months prior to supracondylar osteotomy. The remainder of the procedure, as regards the osteotomy and the period of fixation subsequent to osteotomy, is carried through in the manner already described.

This type of management usually is sufficient to insure a satisfactory result in the posterior dislocation of the hip (Figs. 21, 22, and 23). The only exception to this rule lies in the occasional case which redislocates into an anterior position (secondary anterior dislocation) even after adequate treatment has been carried out (Fig. 37). A discussion of this relatively rare complication will be included in the following discussion of the primary anterior congenital dislocation of the hip.



Fig. 16D The same hip after closed reduction and fixation in plaster spica

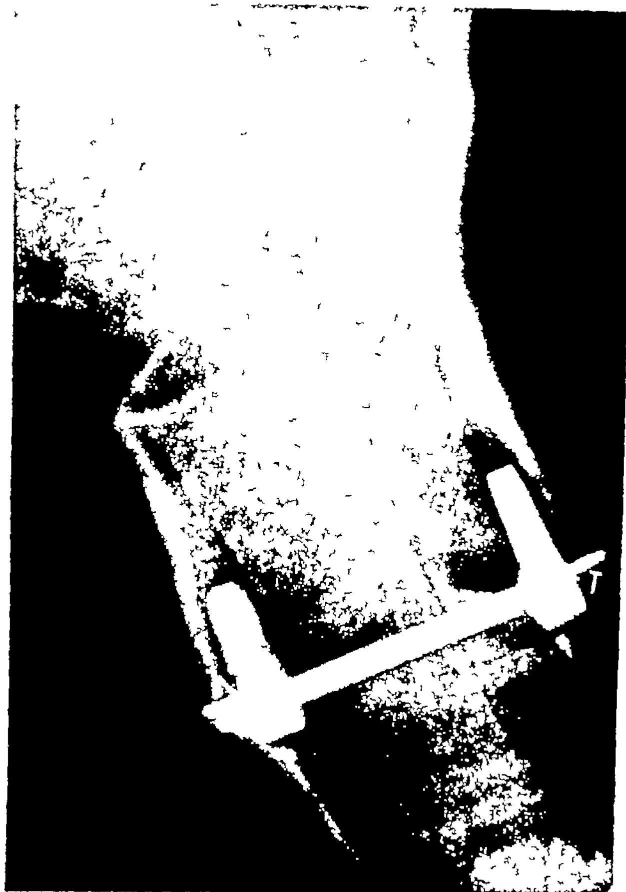


Fig 16E After two months of plaster fixation supracondylar osteotomy was performed for correction of the internal torsion This shows the osteotomy site, the wire through the proximal fragment, with the bow, and reduced position for the hip itself

Treatment of Primary Anterior Congenital Dislocation of the Hip

The existence of a distinct clinical entity of primary anterior congenital dislocation of the hip and the fact that it differs in its response to the usual form of treatment was first called to our attention in 1935. It was noticed that in a high percentage of cases with characteristics of this type, recurrent dislocation of the hip was encountered. The only reference to this in the literature was a case report by Ryerson in 1907. In 1939 a series of 10 cases of primary anterior congenital dislocation of the hip was reported and a form of operative therapy was recommended. This consisted of a massive anterior shelf of bone extending forward to the anterior superior spine in order to produce a shelf of bone above the femoral head whether it was left in an anterior position



Fig 16F Immediately after removal of the final plaster 4 months later Supracondylar derotational osteomy performed at 2 months



Fig 16G. Two weeks later the hip had redislocated into an anterior position. The hip in this view is not seated as deeply as that in Fig. 16A, although it is impossible to tell from the x-ray alone that it has redislocated anteriorly.



Fig 16H. Eight weeks later showing satisfactory reduction restored. During the 8 weeks the hips were again immobilized in a position of abduction and internal rotation This was accomplished by means of long leg plaster casts and connecting cross bars Permanent reduction was maintained following this procedure.

or recurrent anterior dislocation followed (Figs. 24A and 24B) Nine of the ten reported cases were operated in this way (Figs. 25, 26, 27, and 28). In the majority of these, the femoral head was placed in the acetabulum at the time of the shelf operation, but the shelf was extended forward to the anterior superior spine in view of the possibility of a recurrent dislocation Only one of these hips remained in the acetabulum (Fig 29). The remaining



Fig. 16I. One year later showing the improvement in the acetabulum and in the ossification of the femoral head.



Fig. 16J. The same patient 4 years later showing satisfactory reduction and essentially normal joint structures.



Fig. 17A. X-ray showing unilateral posterior congenital dislocation of the hip.

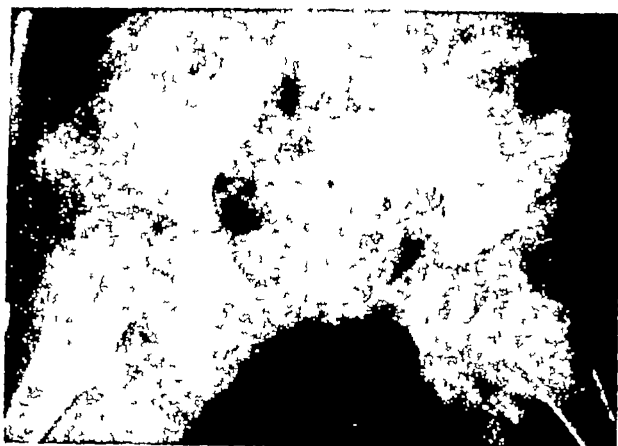


Fig 17B Reduced position of hip following skeletal traction, closed reduction and application of plaster spica



Fig 17D The same hip two years later showing improvement in joint structures.



Fig 17C X-ray of hip and femur 2 months later, after osteotomy for correction of the femoral torsion Internal rotation of proximal fragment maintained (Steinman pin in trochanter This has since been discontinued)



Fig 17E Eight years later showing stable position of the hip although there is some difference in the contour of the head and neck of the femur as compared with the normal side.

eight redislocated into their original, true anterior position (Figs 25, 26, 27, and 28) In the one case which did remain reduced, the shelf was absorbed and eventually disappeared (Fig 29) An entirely satisfactory result was obtained in this patient In the remaining eight patients, results at the time were considered satisfactory except in one instance where an unsatisfactory shelf was obtained As these children grew older, the majority showed shortening of one-half to three-quarters of an inch, and as a result of this shortening, developed a limp and positive Trendelenburg sign Many of them have also developed pain as a result of subsequent traumatic arthritis (Figs



Fig. 18A. X-ray showing bilateral posterior congenital dislocation of the hip. Note the satisfactory appearance of the acetabulum on each side. Treatment for each hip was carried out separately, and was instituted first on the right.



Fig. 18B Four months later after removal of the plaster cast Derotational osteotomy had been performed at 2 months.

25, 26, 27, 28, and 35) Because of this, the massive anterior shelf did not prove to be entirely satisfactory.

The developmental defect in primary anterior congenital dislocation of the hip is a shallow or undeveloped anterior rim of the acetabulum. In the majority of instances the superior rim is entirely satisfactory. The femoral head slips forward through this defect in the anterior margin of the acetabulum to assume its true anterior position. In the recurrent dislocation a similar displacement occurs. Because of this an attempt was made to reconstruct an anterior rim for the acetabu-



Fig 18C. Appearance of both hips immediately after removal of the final cast.



Fig 18D Seven years later showing essentially normal hip joint on each side.



Fig. 19A. Bilateral congenital dislocation of the hip. Note the satisfactory acetabulum on each side.



Fig 19B. After reduction, supracondylar osteotomy and 4 months' plaster fixation for the right hip



Fig. 19D Three years later, showing the satisfactory development of each hip



Fig. 19C Immediately after removal of plaster following reduction and supracondylar osteotomy on the left



Fig 19E. Twelve years later, showing essentially normal hip joints Compare with Fig 19A taken 12 years previously

lum in an effort to maintain the femoral head in a reduced position It proved impossible to raise a leaf of bone from the small bone structure along the anterior margin of the acetabulum in a young child Later an attempt was made to insert an anterior buttress in this type of acetabulum in order to prevent the redislocation of the femoral head at this point (Figs. 34 and 35E) This consists of a large graft removed from the outer table of the iliac crest curved slightly in its lateral plane and driven into a wedge cut along the anterior rim of the acetabulum. The mass of the graft is allowed to project laterally directly in front of the femoral head (Fig 34) This was first attempted by placing the graft outside the joint capsule, but it was impossible to hold the graft in position in this manner Later this was altered to place the graft inside the capsule and suture the capsule over it in an effort to maintain the correct position This is superior to the massive anterior shelf originally described, since in this way the femoral head is maintained in the acetabulum in a normal anatomical position Technically, however, it is a difficult procedure The insertion of a graft perpendicular to the plane of the anterior acetabular rim so that it will project laterally in front of the femoral head is not an easy task. In this type of developmental defect, the femoral head may actually project in front of the anterior rim of the acetabulum even when

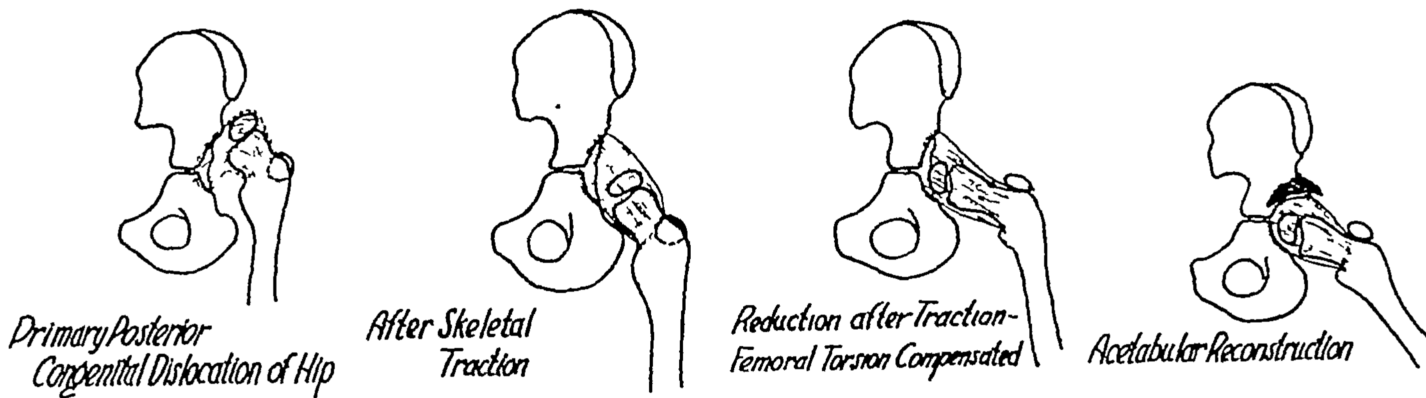


Fig 20A Diagram showing position of unilateral posterior dislocation of hip with an undeveloped acetabulum

Fig. 20B Diagram showing the position after adequate skeletal traction but with the femoral torsion uncorrected.

Fig. 20C This shows the appearance of the hip on correcting the femoral torsion and establishing a satisfactory angle for the femoral neck Note that the superior rim of the acetabulum is inadequate to maintain reduction

Fig 20D. Diagram showing "Shelf Operation," or, in reality an acetabular reconstruction. The superior rim of the acetabulum is simply levered into a horizontal position and supported by bone graft



Fig. 21A. Bilateral posterior congenital dislocation of the hip after application of skeletal traction on the left Note the adequate acetabular depth on the right and inadequate acetabulum on the left.



Fig. 21B. Recurrent dislocation of the left hip after skeletal traction, closed reduction and supracondylar osteotomy. This was due to an inadequate acetabulum.

it is well seated. In these instances the graft is simply laid across the front of the hip joint and sutured inside the capsule. Also, it has been extremely difficult to visualize the anterior buttress by x-ray after operation The portion of the graft which is visualized at times shows no apparent connection with the anterior rim of the acetabulum, but simply appears to lie in the joint opposite the femoral head or femoral neck (Figs 36B and 37D). Whether or not, therefore, the bone graft actually attaches and holds is impossible to state in many instances. An operation of this type may simply serve to produce an anterior ledge for the acetabulum as a result of extensive postoperative scar tissue formation. In either instance, however, the results with the anterior buttress have been quite satisfactory and far superior to those obtained with the massive anterior shelf. It must be stated that these hips have



Fig 21C This shows the hip in plaster immediately after open reduction and shelf operation The extent of the bone graft above the roof of the acetabulum can be seen

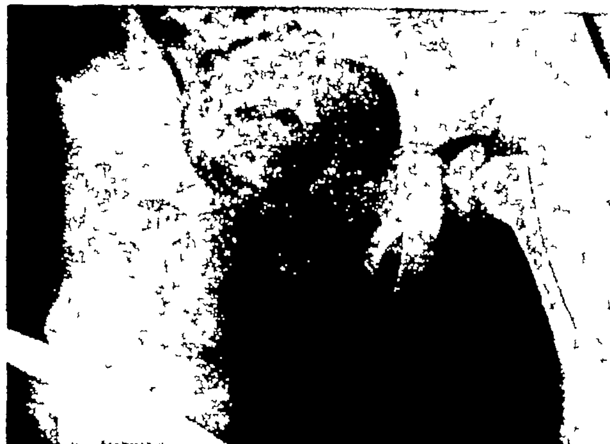


Fig 21E After application of skeletal traction on the right The left hip had been limbered up prior to this



Fig 21D Immediately after removal of the plaster cast A total of 5 months immobilization had followed the shelf operation The projecting portion of this shelf is of no value and will be absorbed



Fig 21F Treatment now completed on both sides Supracondylar derotational osteotomy was also performed on the right



Fig 21G Four years later. Note the improved joint structures on each side

not been followed over a period of years and no end-result studies are available at this time

Open reduction should not be considered or attempted in these patients until after attempted closed reduction has failed to maintain a satisfactory position for the hip In the original report it was stated that open operation was probably necessary in all hips of this type Since then 3 cases of primary anterior congenital dislocation of the hip have held with closed reduction alone followed by derotational osteotomy (Figs. 30 and 31) A total of 25 cases of primary anterior congenital dislocation

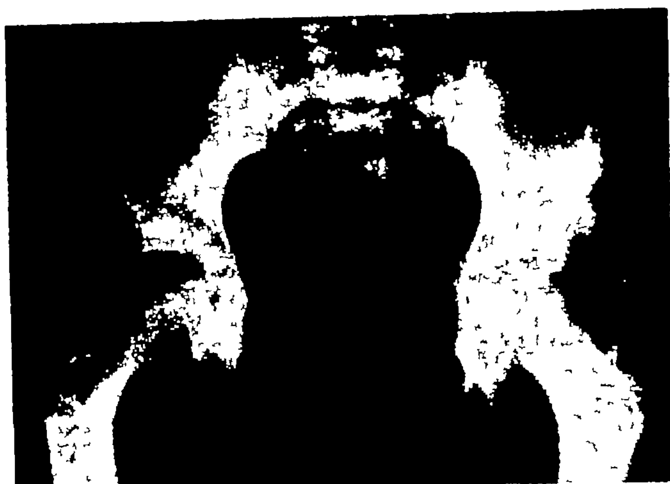


Fig 21H. Eight years later, showing essentially identical contour in the two hips. An adequate acetabulum has been afforded on the left The projected portion of the bone graft which was inserted has almost entirely disappeared.



Fig. 22B After supracondylar derotational osteotomy on the left and 5 months' plaster immobilization, preceded by skeletal traction and shelf operation



Fig 22A. Unilateral posterior congenital dislocation of the hip on the left with an adequate acetabulum An inadequate acetabulum is also present on the right, although the hip is not dislocated.



Fig. 22C Two years later the acetabulum on each side is improving in contour The one on the left is deeper and more adequate than the one on the right

of the hip have been encountered so that in 20 per cent of these cases satisfactory results and normal acatomical relationships have been possible without open reduction of the hip itself. In the original group of 10 cases, one case was maintained in satisfactory position in the acetabulum (Fig. 29), even though shelf operation was performed. Thus a total of 6 cases out of 25 were held in the acetabulum without the influence of surgery even though it was performed in one case. The routine at present for the management of the primary anterior congenital dislocation of the hip is as follows. skeletal traction is instituted in the usual manner and continued for a minimum of three weeks during which time the femoral head is pulled well below the superior rim of the acetabulum. Closed reduction is then attempted, usually without anaesthetic, and the hip is immobilized in a position of abduction and internal rotation, which is necessary in order to place the femoral head in the acetabulum. Immobilization is continued for two months by means of a bilateral hip spica (Fig. 2). Supracondylar derotational osteotomy is then performed and the hip again immobilized in a plaster spica for two months At the end of this time the plaster is removed, and ac-



Fig 22D Nine years later, showing a well-developed acetabular structure on the left. The projecting portion of the bone graft has been almost entirely absorbed The acetabulum on the right is still inadequate and does not afford the protection for the femoral head that is now seen on the left Traumatic arthritis can be expected in the right hip in subsequent years



Fig 23B Following open reduction, shelf operati[on] and supracondylar derotational osteotomy



Fig. 23A Unilateral posterior congenital dislocation of the hip in a child 7 years of age



Fig 23C. Appearance one year later, showing tha[t] the femoral head has displaced upward to imping[e] against the bony shelf

tive use of the extremity is permitted and encouraged If the hip remains well reduced, no further treatment is indicated If, however, a recurrent anterior dislocation follows, as is frequently the case, another attempt at closed reduction is carried out The hip is again immobilized for a period of at least two months. In some instances a plaster spica may not be necessary for this period of fixation Long leg plaster casts connected by crossbars, maintaining a position of wide abduction and internal rotation (Fig 3), will usually suffice. In this way motion of the hip is permitted and i[t] is possible that this motion may help to deepen the acetabulum and insure greater stability Immobilization of this type may be carried ou[t] for several months before resorting to ope[n] reduction If the hip continues to redislocate i[n] spite of adequate conservative treatment, ope[n] reduction is indicated. This is carried ou[t] through the usual anterior approach The join[t] capsule is opened and the interior of the acetabulum is inspected If it is occupied by [a] mass of fibrous tissue, the fibrous tissue i[s] removed in order to deepen the acetabulum a[s] much as possible The anterior rim of the acetabulum is elevated by means of an osteotome. A bone graft is then removed from th[e]



Fig. 23D Eight years later, showing the irregularity in the left hip and lack of normal joint structure Excellent function still exists and the hip remains stable Traumatic arthritis can be expected in this hip, however, in subsequent years. In the older age groups restitution of a normal joint structure by shelf operation is more difficult than it is in younger children Compare with Fig. 22D.

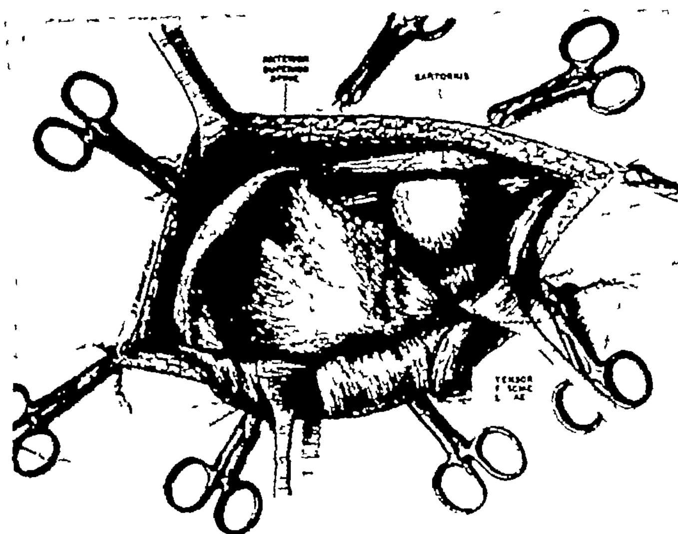


Fig 24A. Drawing which illustrates the position of the femoral head in a primary anterior congenital dislocation of the hip (From J Bone & Joint Surg. 21 648, 1939.)

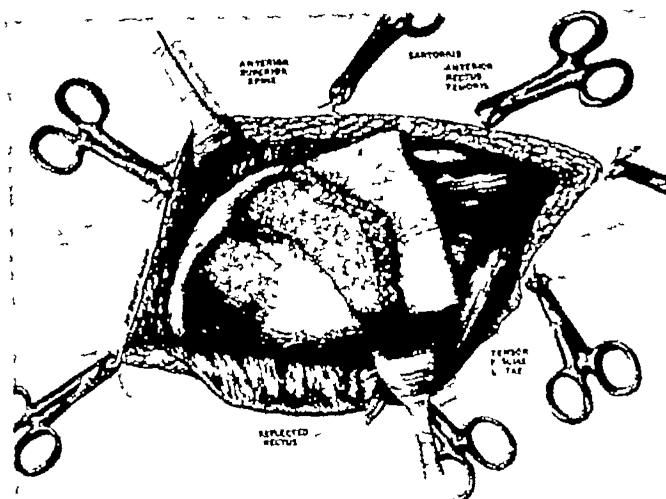


Fig 24B Illustration of the massive anterior shelf which was originally used in these patients in an attempt to insure permanent stability. (From J Bone & Joint Surg. 21 648-664, 1939.)



Fig. 25. Six years after massive anterior shelf with the hip still in an anterior position. There is some irregularity and some arthritic change at the weight bearing surface. This patient had some shortening, a positive Trendelenburg and some discomfort at this time

outer table of the ilium and driven into this defect as well as possible, allowing the body of the graft to project across the front of the hip joint opposite the femoral head (Fig. 34). The joint capsule is then closed outside the bone graft in order to help maintain its position. Bilateral hip spica is then applied, and this position is maintained for three months. Since osteotomy has already corrected the femoral torsion, derotational osteotomy as a rule is not necessary a second time. This fixation is continued for three months after which active use of the extremity is instituted. Only two anterior buttress operations have been performed on the primary anterior type of congenital dislocation of the hip (Figs. 35 and 36). Both of these have shown satisfactory results and have remained well reduced; satisfactory motion and joint function have been restorėd. One of these, number ten in the original report (Fig. 35), had a massive anterior shelf at the age of 18 months. Eight years la-



Fig 26 Another patient with a massive anterior shelf Four years after operation this hip was still dislocated in an anterior position *Unsatisfactory* weight-bearing surface is obvious



Fig 28A Two years after massive anterior shelf with the hip left in its original anterior position



Fig 27 Three years after massive anterior shelf The head of the femur, although appearing to be in the acetabulum, is actually in an extreme anterior position This patient had a slight degree of shortening and a positive Trendelenburg sign



Fig 28B Five years later An entirely unsatisfactory result is obvious This patient had some shortening, a positive Trendelenburg, and some discomfort The femoral head and neck were not developed

ter (Fig 35B), she developed pain, and another open reduction was performed At this operation the femoral head was placed in the acetabulum and an anterior buttress was inserted (Figs 35C, 35D, and 35E) Even though the child is 9 years of age the hip is still well reduced and shows promise of a satisfactory end result (Fig 35E). One hip in this group remained well reduced after open reduction and reconstruction of the superior rim of the acetabulum (Fig 32) Still another case (Fig. 23) had only open reduction, removal of the fibrous tissue from the acetabulum and deepening of the acetabulum by reaming out cartilage and bone from its depth This hip, although it has remained reduced, shows definite evidence of traumatic arthritis (Fig. 33G)

An occasional posterior dislocation of the hip redislocates in an anterior position as a result of a defective anterior acetabular rim This may occur even after shelf operation (Fig 37) In the event this does occur the treatment becomes identical with that of the primary anterior type Immobilization in internal rotation and abduction is again carried out and in the event this fails open reduction and anterior buttress operation are indicated (Fig 37)



Fig 29. X-ray showing satisfactory position of hip 8 years after massive anterior shelf was performed. This represents the one case in the original group which remained reduced. The massive anterior shelf, since it has not been used, has now been entirely absorbed



Fig. 30A. X-ray showing unilateral primary anterior congenital dislocation of the hip.



Fig. 30B. After closed reduction and fixation by means of long leg plaster casts and connecting cross bars.

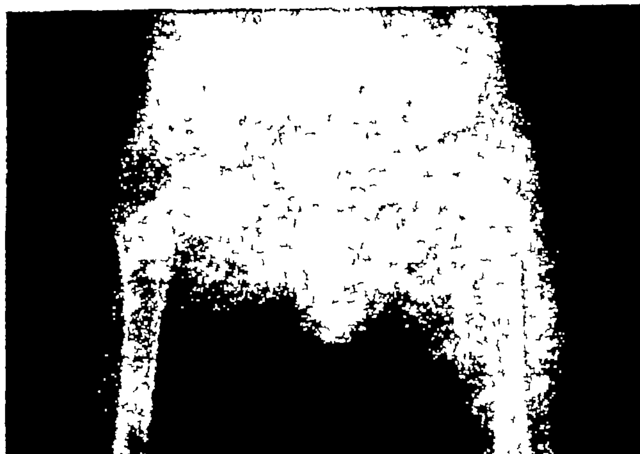


Fig 30C Six months after removal of the plaster. Supracondylar derotational osteotomy had been performed The acetabular roof appears to be questionable at this time.



Fig. 30D Two years later the hip is still well reduced and acetabulum is improving in appearance.



Fig. 30E. Three years later. Reduction is still maintained and the acetabulum now appears quite adequate. This illustrates how a satisfactory result can be obtained in some primary anterior dislocations by means of closed reduction.



Fig 31A X-ray showing unilateral primary anterior congenital dislocation of the hip



Fig 31B. Three years after closed reduction and supracondylar osteotomy, showing satisfactory position of the hip and improved appearance of the joint structures. This illustrates again that closed reduction is occasionally successful in a primary anterior congenital dislocation



Fig 32A. Unilateral primary anterior congenital dislocation of the hip with a totally inadequate acetabulum.



Fig 32B. Immediately after removal of plaster showing the femoral head well seated beneath the shelf Supracondylar osteotomy was performed



Fig. 32C Five years later Reduction has been maintained but some irregularity is evident in the overlying shelf.



Fig. 33A Unilateral primary anterior congenital dislocation of the hip with a totally inadequate acetabulum



Fig 33B Appearance following skeletal traction, closed reduction, and supracondylar derotational osteotomy.



Fig. 33C One year later, showing recurrence of anterior dislocation.



Fig. 33D. Immediately after second closed reduction and plaster fixation

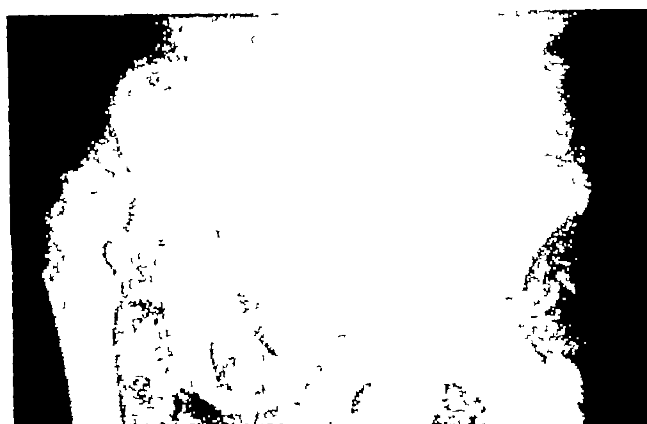


Fig. 33E. Three years later. Anterior dislocation has again recurred



Fig. 33F. Showing appearance following open reduction and deepening of the acetabulum by reaming out its depth. The articular cartilage of the femoral head was placed directly against the denuded depth of the acetabulum.



Fig 33G Three years after open reduction and six years after institution of treatment. The femoral head remains in the acetabulum, which is irregular in contour and inadequate in depth The hip already has some evidence of traumatic arthritis which will undoubtedly increase with age.

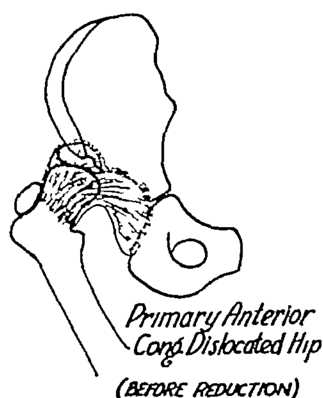


Fig 34A Diagram showing primary anterior congenital dislocation of the hip

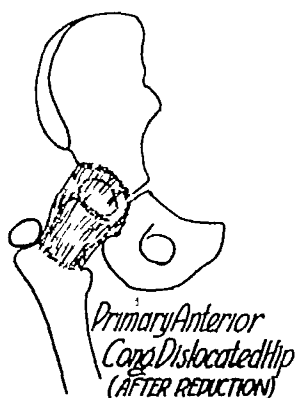


Fig 34B Immediately after reduction and supracondylar derotational osteotomy which were preceded by skeletal traction

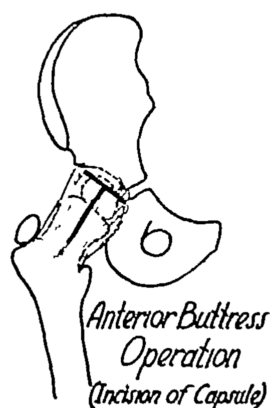


Fig 34C Diagram showing type of incision which is made in the joint capsule for insertion of a bone graft to be used as an anterior traction

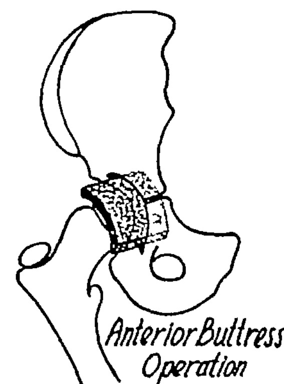


Fig 34D Diagram showing the anterior buttress inserted The joint capsule is then closed outside this graft in order to maintain its position



Fig 35A Primary anterior congenital dislocation of hip. Three years following massive anterior shelf operation, the hip is still dislocated in an anterior position There were no symptoms at this time



Fig 35B Eight years after the anterior shelf was performed, the patient had developed pain in this hip Some shortening and a positive Trendelenburg sign were still present

Treatment of Simple Upward Subluxation of the Hip

This type of dislocation, as stated previously, is entirely the result of an inadequate superior rim of the acetabulum which permits the femoral head to displace upward as a result of joint motion, muscle pull, and weight bearing Open reduction and reconstruction of a superior acetabular rim is essential in all cases of this type if stability of the hip is to be maintained Treatment, therefore, consists of preliminary skeletal traction in order to bring the femoral head well below the superior rim of the acetabulum. The open reduction and shelf operation are then performed The shelf operation actually consists of an acetabular reconstruction in which the superior rim of the acetabulum is levered down into a horizontal position and anchored there by means of a bone graft wedged between it and the body of the ilium (Figs 38 and 39). The interior of the acetabulum should be inspected at the time of



Fig 35C. After open reduction in which the femoral head was placed in the acetabulum and an anterior buttress was inserted The bone graft anterior to the femoral head is difficult to outline through the plaster cast.



Fig. 35D Immediately after removal of the plaster. The anterior buttress can be easily seen



Fig. 35E Nine months after operation. The anterior buttress is still holding the femoral head in the acetabulum. The buttress is attached to the anterior acetabular rim. The superior portion of this acetabulum appears to be questionable.



Fig 36A Primary anterior congenital dislocation of the hip.



Fig 36B One year later. Reduction has been maintained but very little evidence of the anterior bone graft can be seen. The superior rim of this acetabulum was reconstructed at the same time.



Fig 37A. Primary posterior congenital dislocation of the hip which after adequate treatment redislocated anteriorly. Inadequate superior rim of acetabulum is obvious Initial treatment consisted of skeletal traction followed by shelf operation.



Fig 37B This shows the adequate superior rim of the acetabulum immediately after the plaster was removed



Fig 37C. One year later the hip was redislocated anteriorly



Fig 37D Following open reduction and insertion of bone graft and anterior buttress The bone graft is not attached to the anterior rim of the acetabulum but instead is attached to the anterior surface of the femoral neck The graft is outlined



Fig 37E One year after the anterior buttress operation Reduction has been maintained even though the buttress is not attached to the anterior rim of the acetabulum The position of the buttress is outlined The buttress is obviously not maintaining the reduced position but the scar anteriorly, resulting from the surgery, may be sufficient to do this.



Fig 38A Typical picture of a simple upward subluxation resulting from inadequate superior rim of the acetabulum



Fig 38B Immediately after open reduction and shelf operation



Fig 38C After removal of the plaster cast



Fig. 39A. X-ray of another simple upward subluxation



Fig 38D Two years later The joint structure appears improved and the concavity of the acetabulum is being established.



Fig. 39B Six years following reduction by means of skeletal traction, shelf operation, and supracondylar derotational osteotomy An adequate weight-bearing surface has been afforded although some irregularity persists



Fig. 38E Eight years later Smooth acetabular surface is seen Normal joint motion and normal function are present at this time

operation if there is any question about the femoral head being well seated in the acetabulum Plaster fixation is then continued for three months. Femoral torsion may at times be present in hips of this type, but may also be entirely absent The necessity of supracondylar derotational osteotomy, therefore, depends on the demonstration of the presence of femoral torsion at the time of open reduction. If a position of marked internal rotation of the extremity is necessary in order to adequately seat the femoral head in the acetabulum, supracondylar derotational osteotomy should be performed Plaster fixation following the shelf operation is continued for three months. If derotational osteotomy is necessary it is continued for another period of two months before activity in the hip is begun.

Summary

Treatment of congenital dislocation of the hip from the time of birth to the age of eight years has been discussed Classification into the three clinical types of dislocation is also included with a discussion of treatment usually required for each type. Adequate treatment in this age group will usually insure restoration of normal anatomical relationships and permanent maintenance of satisfactory joint function

THE OPERATIVE TREATMENT OF CONGENITAL DISLOCATION OF THE HIP INDICATIONS AND METHODS

A Bruce Gill, M D.

In treating congenital dislocation of the hip, it must always be borne in mind that the efficacy of each and every method to restore a normal, or perfect, hip is absolutely dependent, first, upon the nature and the degree of the pathological changes that have taken place in the structures of the hip prior to the beginning of treatment, and, second, upon the ability of these structures to return to the normal during the period of growth subsequent to the reduction of the dislocation

There are some dislocations, in which the primary or the secondary pathological alterations of structure are marked, that cannot be reduced either by bloodless or by operative methods For these hips only palliative operations are available (Figs 1, 32, and 33)

Excluding the severe foetal or embyrological dislocations, which comprise possibly 4 to 6 per cent of all dislocations and which are irreducible even at birth, approximately 80 to 85 per cent of the remainder can be reduced bloodlessly, i.e , the head of the femur can be brought into approximation with the acetabulum, if treatment is instituted early and before secondary pathological changes of structure prevent reposition. The other 15 to 20 per cent of the reducible hips must be replaced by open operation



Fig 1 R T 5 14 22 Marked Congenital deformities present at birth Bilateral dislocation of hips, defect of right femur, deformities of hands and feet

Of the hips which have been reduced bloodlessly only approximately 15 to 35 per cent (an exact percentage cannot be stated) will of themselves develop into normal hips during the process of growth. Complete or partial luxation occurs in all others, two-thirds during the first two years after reduction and the remaining third later.

Surgical operation is indicated (1) to reduce dislocations that cannot be reduced bloodlessly, (2) to reconstruct hips that have become luxated or subluxated subsequent to bloodless or open reduction, (3) to reduce the disability in irreducible dislocations

The pathological anatomy and not the age of the patient is the final and absolute criterion that determines treatment of congenital dislocation of the hip. During the prewalking age a child may have merely a predislocation, or a complete dislocation that can be reduced bloodlessly or only be open operation, or it may have a hopelessly irreducible dislocation. Also, the same type of operation may be required in early years as during the period of adolescence It must be emphasized, however, that age is a determining factor in the production of the secondary pathological changes that occur in the structures of the hip joint

subsequent to dislocation. Therefore, the earlier a predislocation, a subluxation or a frank dislocation is discovered and treated the less marked have the secondary alterations become and the greater the opportunity for nature to resume the interrupted development of the structures of the hip. Abnormality of the hip should be recognized at birth or soon thereafter.

I. Open Reduction when Bloodless Reduction is Impossible (Figs. 2 and 3)

At the time of operation one can always observe the obstacles which made bloodless reduction impossible or incomplete. They consist of abnormalities of the acetabulum, of the head and neck of the femur, and of the capsule. In addition, a shortening of pelvi-femoral muscles (the adductors) may impede reduction. The technic of the operation which must be used to secure reduction in any particular case, therefore, is dependent upon the pathological anatomy which is found to be present.

Preliminary traction is useful in those cases in which during the attempt to reduce the hip by manipulation, it was not possible to bring the head of the femur down to the level of the acetabulum, and likewise, in those cases over four years of age in which the head is situated well above the socket[1] (Figs. 4 and 5).



Fig 3 J. P (Same case as in Fig 2) 10 12 45 Eight months after open reduction, it remains reduced Acetabular root is developing (Gill, A B.. Nelson's Loose-Leaf Surgery, 1946)



Fig 2 J P female, age 4 years (Dec, 8, 1944) Dislocation of left hip. Small, shallow, deformed acetabulum Note the acetabulum which is an extension upward of the primary one, small conical, incompletely calcified head, deformed neck Two attempts to reduce bloodlessly failed (An attempt two years earlier by another surgeon had failed) Reduced 2 2 45 by open operation (Gill, A B Nelson's Loose-Leaf Surgery, 1946)



Fig. 4 E.S.G., 10 years of age. High dislocation, secondary deformity (Gill, A. B · J Bone & Joint Surg. 17 1, 1935)



Fig 5 E C (Same case as in Fig 4) Shortening has been overcome by gradual prolonged traction followed by shelf operation which was performed six weeks ago (Gill, A B J Bone & Joint Surg 17 1, 1935)

Technic of Operations that Accomplish Reduction of the Hip, i e, Replacement of the Head of the Femur within the Acetabulum

A modified Smith-Peterson incision is employed whereby its anterior portion is curved downward and backward from the anterior superior spine of the ilium in the manner of the Ollier incision This is essential in certain cases in which it is necessary to expose the great trochanter and the base of the neck of the femur by carrying the incision backward to a point two or three inches below the top of the trochanter

The flap of skin and subcutaneous tissue is dissected downward and backward, disclosing the gluteal fascia The crest of the ilium is identified In younger individuals it consists largely, or altogether, of cartilage, depending on the age of the child With a scalpel the cartilage is cut from the underlying bone, is reflected upward, carrying the attached abdominal muscles, and is displaced sufficiently inward to expose the bony crest of the ilium from the anterior superior spine to slightly beyond the thickened portion of the crest which is known as the tubercle. The tensor fascia femoris and gluteus medius muscles are then detached subperiosteally from their origin by sharp dissection with scalpel or sharp osteotome. The muscles can then be stripped subperiosteally from the ilium with the handle of the scalpel. The interspace between the sartorius and the tensor fascia femoris is then identified and these muscles are separated by blunt dissection with the fingers or the handle of the scalpel With the blade of the knife the tensor fascia femoris and the anterior origin of the gluteus minimus are then freed subperiosteally from the anterior margin of the ilium down to the anterior inferior spine

We have now reached an important point in our dissection We have gone as far as we can by blunt subperiosteal stripping and have not yet reached the acetabular cavity which lies a half-inch to an inch or more beyond this point. The periosteum is intimately attached to the bone The capsule blends with the periosteum above the acetabulum The posterior origin of the rectus femoris and some fibers of the gluteus minimus are attached to the capsule. With a sharp thin-bladed osteotome the ilium is stripped of all these structures until the rim of the acetabulum is reached from below the anterior inferior spine backward The rim of the acetabulum can usually be identified with the finger, but to make certain of it, a sharp-pointed forceps should be passed over it into the acetabular cavity, or the stripping with the osteotome should be continued until it has made a small opening into the cavity, whence joint fluid will appear in the wound With the osteotome or with a scalpel the muscles are freed from the capsule and pushed downward and backward. The white glistening capsule is thus exposed freely, and the head of the femur can be identified within it.

After doing whatever operation may be necessary on the hip, the wound should be closed be retracting the cartilage of the iliac crest into its normal position and by suturing the gluteal muscles firmly to it. If the muscles are not restored to their original point of origin their future function will be impaired.

After the hip joint has been exposed by the method described above, the surgeon must do whatever may be necessary to secure and maintain reduction.

1) It may be possible to replace the head within the acetabulum by manipulation. If it goes deeply into the socket and is stable (i.e., resists upward thrust of the femur) in a position of moderate abduction, it may thenceforward be treated in the same manner as a case of bloodless reduction These cases are rare. The adhesion of the capsule to the side of the ilium is not the chief obstacle that prevents primary bloodless reduction.

If secure reduction cannot be accomplished in this manner the capsule must be opened by detaching it from the upper margin of the acetabulum and by a second longitudinal incision at a right angle from the center of the first one. This permits free exposure of the joint. At the conclusion of the operation the two flaps of the capsule made by the longitudinal incision are sutured together. Adhesion of the capsule to the margin of the acetabulum will occur during healing of the wound

From this point the surgical procedures will depend upon the morbid anatomy of the hip.

2) Soft tissues, namely, an elongated and thickened ligamentum teres and fatty fibrous tissue which probably arises from the Haversian gland, prevent deep replacement of the head within the socket. They must be excised with scalpel and curved scissors.

3) After these soft tissues are excised it may be found that the bony acetabulum is too small to receive the entire head. With a sharp curved gouge enough of the acetabular cartilage may be removed to afford sufficient room.

4) If the head rides too high and cannot be placed easily in the socket, it should not be forced into it lest bony ankylosis or at least marked limitation of motion result. The roof of the acetabulum must be enlarged upward with a curved gouge until the head slips easily into place. The new acetabular roof should be rounded to fit the head and it should not be oblique lest the head slip readily out from beneath it.

5) If, after any of the above procedures, the socket is too shallow, the head of the femur projects too far beyond its margin, and the stability is impaired, a shelf or buttress operation should be done to make a competent acetabulum. This operation will be described later.

6) The neck of the femur is short, the head sometimes being almost sessile upon the shaft. The capsular cuff attached to the short neck and the great trochanter prevent entrance of the head into the acetabulum even when the acetabulum is large enough to receive the head Sometimes the capsule is attached to the head at the margin of the articular cartilage. The author has observed bands of fibrous tissue passing from the capsule to near the top of the head It is often impossible, also, to bring the head forward to the acetabulum. This type of case is fairly common.

These hips cannot be reduced until the capsule has been stripped from its femoral attachment. It may also be necessary to lengthen the neck by transplanting the great trochanter farther down on the shaft of the femur.

7) Deformities of the head of the femur are very common in old dislocations but are not so frequently seen in children under four years of age and, as a rule, do not prevent reduction in these latter cases. Flattening of the head where it has been resting on the side of the pelvis, conical or pear-shaped structure, possibly due to defective and irregular development of the capital epiphysis, wart-like excrescences, and incomplete and irregular growth of the articular cartilage are secondary pathological changes which occur because of prolonged dislocation.

8) Anteversion of the neck does not interfere with reduction, but in order to maintain reduction the extremity must be held in internal rotation. The author has observed this condition frequently in those cases where the neck of the femur is short.

9) Hour-glass constriction of the capsule is a secondary development which is seen in old dislocations. Sometimes the inferior portion of the capsule has been drawn upward across the acetabulum and has become adherent to its upper margin like a drumhead and completely separates the acetabular cavity from the capsular sac which contains the head. These are conditions which are found only in old irreducible dislocations.

10) A comparatively rare condition, and one which does not prevent bloodless or open reduction, is delayed calcification of the neck of the femur. There is reason to believe that congenital coxa vara may be explained by this phenomenon. All of the author's cases have had a concomitant shortening of the femur. (Figs. 6 & 7).

11. Thickening of the capsule and shorten-



Fig 6 J K, female, one year of age 9 18 45 Had bloodless reduction when four months of age Hip remains reduced Femoral head is visible Neck invisible in x-ray Congenital shortening of the femur Apparent coxa vara

ing of the pelvi-femoral muscles are observed in the old dislocations.

It must be borne in mind that if the head of the femur cannot be placed in the primary acetabulum the operation must be included in the group of palliative operations for irreducible dislocations.

II. Reconstruction of Hips that Luxate or Subluxate Subsequent to Bloodless or Open Reduction

Approximately 80 to 85 per cent of all dislocations in children up to four or five years of age can be reduced bloodlessly. The earlier reduction is attempted the more easily is it accomplished and the better are the changes for permanent reduction. But only from 15 to 35 per cent of this group will develop into normal hips during the period of growth. The remainder will become luxated or subluxated during the succeeding years, most of them during the first two years. These hips, therefore, constitute the largest and the most important field for operative surgery We should have definite views concerning the indications for surgical intervention and the type and technique of the most useful operations



Fig 7 J K (Same case as Fig 6) 10 14 40 Hip remains reduced Neck of femur has become calcified Marked coxa vara Delayed calcification of neck may be explanation of congenital coxa vara

Indications for Operation

There are some general principles that must be kept always in mind (1) The dislocation should not be considered to be "cured" until there has been a restoration of normal structure as well as of normal function This restoration may occur as early as several years after reduction or may be delayed until the age of puberty (2) All reduced dislocations, therefore, should be kept under regular observation until the underlying dysplasias of the head and the acetabulum have been corrected by growth processes (3) Since at least 65 per cent of reduced hips, according to our present knowledge, will not of themselves become normal it must be determined at what time and for what conditions surgical intervention is indicated. (4) All operated hips,



Fig 8 M M., female, nineteen months of age (Gill, A. B. J Bone & Joint Surg 18 4, 1936)

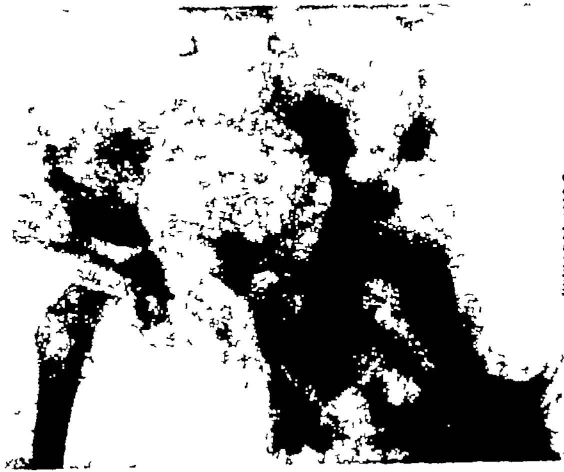


Fig 9 M M (Same case as Fig 8.) Ten months after bloodless reduction (Gill, A B · J. Bone & Joint Surg 18 4, 1936)

also, should be kept under observation for as many years as possible in order to determine the ultimate result from the operation. Here also we may predict that only restoration of normal anatomy will guarantee future freedom from symptoms and that the method of operation which secures the nearest approach to this normal will be the most efficient in the long run.

1) If the head of the femur slips out of the socket soon after fixation casts or braces have been removed, open operation by methods described in the preceding section of this paper is indicated (Figs. 8, 9, 10, and 11). Anatomical defects which made complete bloodless reduction impossible are revealed at the operation. My own statistics, which may not conform to those of other surgeons, show that 28.5 per cent became redislocated within the first year after reduction. Even retention in casts for as long as eighteen months did not prevent dislocation at the end of that period. Those hips that remain stable at the end of four to six months of fixation have the best ultimate prognosis.

2) Hips that remain reduced should be kept under close observation and x-rayed at fre-



Fig. 10 M M (Same case as Fig. 8.) One year and three months after reduction Hip has been fixed in plaster cast this entire period Illustrates principle that if head does not remain in acetabulum after four months' fixation it may not do so even after prolonged fixation in plaster (Gill, A B J. Bone & Joint Surg 18 4, 1936)

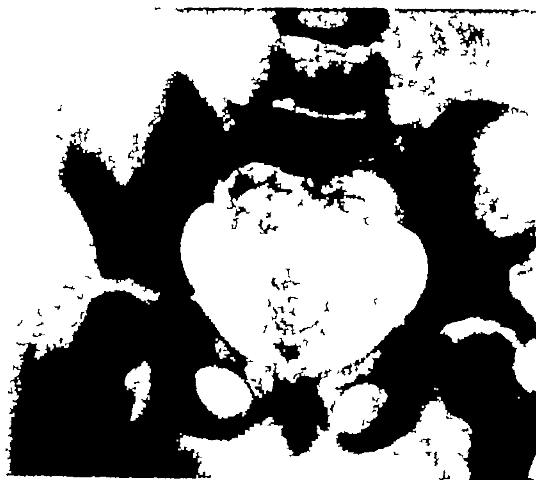


Fig 11 M M (Same case as Fig 8) Eight months after reconstruction of acetabulum (Gill, A B.· J. Bone and Joint Surg 18 4, 1936)

quent intervals during the early years and at longer intervals during later years Subluxations and later complete luxations occur chiefly because the acetabular roof does not develop normally The x-rays may show for an indefinite length of time that the bony roof is poorly developed and oblique and that the head stands out *from the floor so that* Waldenstrom's oval is absent or is small, but the head is not migrating upward. Operation is not indicated (Figs 12, 13, 14, 15, 16) Some of these hips will eventually become normal by natural growth processes But as soon as the head begins to migrate upward, the shelf, or buttress, operation is indicated (Figs 17, 18, 19, 20) *The author makes an exception to this* rule in children under five or six years of age



Fig 12 R M C, female, 5 27 35 Seven years after reduction in 1928 when three years of age Dysplasia of acetabulum still apparent Head displaced slightly outward but no upward displacement Operation not indicated (Gill, A B J Bone & Joint Surg 25 1, 1943)



Fig 14 J M, female Two and a half years of age Bloodless reduction on 3 7 32 (Gill, A B J Bone & Joint Surg 25 1, 1943)



Fig 13 R M. C (Same case as Fig 12) 4 25 42 Fourteen years after reduction Perfect hip Seventeen years of age (Gill, A B J. Bone & Joint Surg. 25 1, 1943)



Fig 15 J M (Same case as Fig 14) 5 27 35 Three years after reduction Continued dysplasia of acetabulum and head of femur Head of femur is not being displaced upward Operation is not indicated (Gill, A B J Bone & Joint Surg 25 1, 1943)



Fig 16 J M (Same case as Fig 14) 4 25 42 Ten years after reduction Twelve years of age Perfect hip (Gill, A. B. J Bone & Joint Surg 25 1, 1943)



Fig 18 I R (Same case as Fig 17) Ten years after reduction Marked subluxation Has developed pain and limp Reconstruction operation performed 5 20 40 (Gill, A B J Bone & Joint Surg 25 1, 1943)



Fig 17 I R, female Four and one-half years after reduction, 3 15 29, when four years of age Dysplasia of head and acetabulum Moderate subluxation outward and upward displacement of head No symptoms Operation indicated (Gill, A B, J Bone & Joint Surg 25 1, 1943)

He applied an abduction brace which keeps the head pointed into the floor of the socket and prevents upward displacement. The very few cases in which the shelf has melted away were in children under five years of age. If after the brace is removed the head again rides upward, operation should be performed. It should also be remembered that upward subluxation may not become evident for some years after the primary reduction and also that it may be present for many years without causing symptoms of fatigue, pain, and a positive Trendelenburg sign. However, the onset of these symptoms is evidence of instability of the hip and the necessity for operation.

The most important field for the shelf operation is in the cases of subluxation, although it is useful also in selected cases of old, irremedial dislocation. In the former cases the head of the femur is still in the primary acetabulum. What moderate upward displacement there may be can be corrected at the time of operation or, if necessary, by preliminary traction A hip that is normal or very closely approaching it may be expected as an end result.

Technic of the Buttress Operation in Subluxation

The approach to the hip joint has been fully described on a preceding page The capsule



Fig 20 I R (Same case as Fig 17) 3 30 42 Two years after operation Perfect result from operation Normal function. (Gill, A B J Bone & Joint Surg 25 1, 1943)

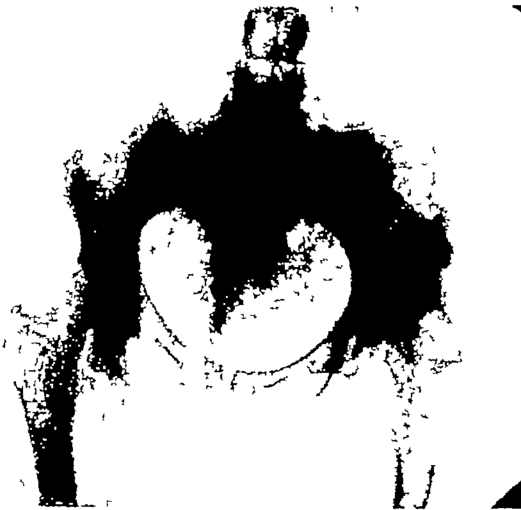


Fig 19 I R (Same case as Fig 17) 8 4 40 Three months after operation Showing method of buttress construction (Gill, A B J Bone & Joint Surg 25 1, 1943)

with the underlying periosteum of the ilium has been stripped completely from the ilium to the very margin of the acetabular rim Muscles have been stripped from their capsular attachment The acetabular rim has been absolutely identified by thrusting an instrument over it into the acetabular cavity.

If the capsule is thick, it is pared off with a scalpel or a sharp osteotome to reduce the thickness The portion of capsule which was stripped from the ilium and which lies as a wedge-shaped mass just below the acetabular rim must be cut away. The shelf to be constructed must lie in close approximation to the head of the femur and be separated from it only by a thin layer of capsule This part of the capsule which lies in the hip joint after the operation is completed probably undergoes later absorption. If it is thick, the head of the femur will move upward as absorption takes place

Next, a bone flap is outlined on the side of the ilium It must be sufficiently wide to cover the head of the femur. The upper margin is curved in a line paralleling the acetabular rim The base of the flap is immediately above the rim The anterior border of the flap is at the anterior margin of the ilium. It extends far enough backward to envelop the entire upper portion of the head posteriorly It consists of the outer table of the ilium. With a thin-bladed sharp osteotome this flap is then slowly and gradually detached from the ilium Care is taken not to break it. As one proceeds with the separation of the flap, frequent movements of the osteotome produce slight green-stick fractures of it so that the surface of the flap becomes curved to fit the rounded head of the femur During the detaching of the flap the osteotome is always held flat against the side of the ilium except for the slight movements to produce green-stick fracturing

As one approaches to about three quarters of an inch from the acetabulur rim, the handle of the osteotome is gradually carried farther and farther from the side of the pelvis, and the instrument is driven in deeper and deeper and parallel to the acetabular roof The cutting edge of the osteotome now has penetrated

just above the roof of a depth of an inch and a quarter, or more, depending on the size of the patient A broader bladed osteotome is now substituted for the narrower one and the entire acetabular roof is pried downward. The bone flap turned down from the side of the ilium is now a smooth continuous extension of the roof. There is now a deep wedge-shaped cleft in the ilium above the detached roof.

Now one prepares the bone wedges from the crest and the side of the ilium below the crest. The author, however, usually prepares the wedges before detaching the flap and reflecting the roof downward, lest I or my assistant accidentally catch a piece of gauze on the bone-flap and tear it off while preparing the wedges The wedges consist of almost the full thickness of the crest and the outer table of the ilium and the cancellous bone between the outer and inner tables. The thickest, longest wedge is that obtained from the tubercle of the crest The ones removed from the crest anterior to the tubercles are naturally thinner. The thick one is one and a half to two inches in length The thinner one or two removed from the anterior part of the crest is shorter.

Now to return to the acetabulum. An assistant makes traction on the leg and pulls the head of the femur down as far as possible. It can be seen and felt to move downward. Preliminary traction preceding operation may have been necessary in older patients to bring the head fully down to its normal position. The acetabular roof is then pried farther downward until it rests firmly against the head. The bone flap is bent further downward until it is in complete approximation with the head. While the osteotome remains within the cleft, the assistant pushes up on the leg, but the head of the femur does not now move upward Replace the osteotome with the bone wedges and the femur can never again be displaced upward The heavy, long wedge is driven in first with an impactor as deeply as it will go, an inch and a half to two inches above the reflected roof. It projects slightly beyond the external surface of the ilium. A second, shorter, and thinner wedge is then driven in below the first one. It cannot enter far, but it is deep enough to be fixed securely in place. The thick portion of the wedge projects above the bone flap and forces it down upon the head Frequently, a third, still shorter wedge is driven in beneath the second wedge, projecting be-



Fig. 21 J D 6 2.39. Seventeen years after reduction. Obliquity of acetabulum, subluxation Satisfactory Operation advised but declined because of complete absence of symptoms (Gill, A B J Bone & Joint Surg 25 1, 1943)



Fig 22. J D. (Same case as Fig. 21.) 1.29 42. For twenty years had satisfactory reduction with complete absence of symptoms Recently has developed tiredness and limp after walking an unusual distance. Operation 4.23 42. Demonstrates that anatomical imperfection of hip eventually produces symptoms. (Gill, A. B. J Bone & Joint Surg. 25 1, 1943.)

yond it This wedge projects to the very margin of the flap, reinforces it and rounds it more above the head One can now see why the author prefers to call this a buttress instead of a shelf operation Additional small pieces of bone from the side of the ilium, like bone mush or bone cement, may be used to fill in any interstices there may be between the wedges and in front or behind them If, by any mishap, the bone flap has been broken off at its base, no harm has been done. It is placed in its appropriate position as the wedges are driven in and is held by them firm and curved over the head of the femur The flap, the reflected roof, and the wedges are soon fused into a solid mass of bone with the body of the ilium. If the outer margin of the flap extends too far beyond the head of the femur, the unnecessary portion will in time be absorbed because it has no function. Now, to satisfy yourself that your job has been well done, push the leg firmly upward, try to pull it down, there is no movement of the head.

Close the wound as described in the earlier part of this paper Apply a plaster cast far eight to twelve weeks Allow the patient to walk by the end of three months, when solid fusion of the buttress has taken place All movements of the hip are normal, the hip is stable and painless, the Trendelenburg sign is negative with the exception of older cases in whom irregularity of the head of the femur, such as flattening, causes limitation of rotation (Illustrations showing technique of "buttress operation," Figs 11, 19, 23, 24, 25, 26, and 27)



Fig 23 J D (Same case as Fig 21)6 25 42 Two months after operation Shows method of construction of 'shelf" without opening the capsule (Gill, A B J Bone & Joint Surg 25 1, 1943)



Fig. 24 H T , male, forty-two years of age (July 24, 1944) Subluxation of left hip He had a bloodless reduction when two years of age, has had increasing pain, tiredness, and disability during the past ten years Negative Trendelenburg sign (Gill, A B , Nelson's Loose-Leaf Surg 1946.)

The Trendelenburg sign may be positive following a buttress operation in old luxated hips because the floor of the acetabulum has been filled up with bone and the head of the femur lies too far from the mid-line of the body. I have never observed a positive Trendelenburg sign following the type of operation which I have described above

III Irreducible Dislocations

The dislocation is irreducible when the head cannot be replaced in the primary acetabulum. These hips cannot by any means become normal hips, but much may be accomplished to improve stability and even to restore it completely and at the same time retain partial motion

1 Hips That Are Irreducible at Birth

These dislocations constitute but a small percentage of total dislocations. In the author's experience they are always bilateral The fe-



Fig 25 H T (Same case as Fig 24) 7 23 45 One year after "Shelf" (Buttress) operation Complete relief from symptoms (Gill, A B Nelson's Loose-Leaf Surg 1946)



Fig 27 V P , female Showing result four years after buttress operation Perfect result Normal function



Fig 26 H T. (Same case as Fig 24) 7 23 45 Showing freedom of abduction (Gill, A B Nelson's Loose-Leaf Surg 1946)

moral heads lie high on the pelvis in partially formed secondary acetabula They are so rigid that even under general anesthesia no longitudinal motion can be obtained. The perineum is very broad and the adductor muscles are at times enlarged, dense, and unyielding. Other deformities, club feet, club hands, hyperextension of knees, rigidity of joints, are present and are accompanied frequently by mental retardation

Some authors believe that these hips represent embryological dislocations and even go so far as to assert that the upper end of the femur never was in approximation with the primary acetabulum. The author agrees that they occur very early in the life of the individual but is unable to comprehend how the upper end of the femur can originate above and independent of the primary acetabulum since both are formed from a common embryological structure.

Nothing can be done for these hips. Other deformities of the lower extremities should be corrected as far as possible so that the child may be able to stand on its feet, if or when it learns to assume the upright position.

2 Reconstructions of the Hip-Joint at a Site above the Primary Acetabulum (Figs 28, 29, 30, and 31)

The head of the femur can sometimes be pulled down on the pelvis by preliminary traction.



Fig 28 J V , female, 12 years of age (Gill, A B , J Bone & Joint Surg 17 1, 1935)

At the time of operation the capsule is opened, the head is delivered from the capsule and is brought forward to the anterior margin of the pelvis The primary acetabulum is then enlarged upward until the head can be placed in it gently and without force It remains in this acetabulum when the leg is moderately abducted but slips out of it when the leg is brought into a longitudinal position because the acetabular roof is not sufficiently large to cover the entire head even though it is not oblique A bone shelf or buttress is then constructed to cover the entire head, much in the manner already described, with the exception that the bone wedges cannot be driven in so deeply As the buttress is constructed an assistant holds the leg in sufficient abduction to prevent redisplacement of the head This abduction is maintained constantly until the cast has been applied

When the head of the femur lies higher on the side of the pelvis where the ilium is thin-



Fig 29 J B (Same case as Fig 28) Shortening partially reduced by traction Operation Six weeks after operation (Gill, A B , J Bone & Joint Surg 17 1, 1935)



Fig 30 A L , female, adult (Gill, A B J Bone & Joint Surg 17 1, 1935)



Fig. 31 A L (Same case as Fig 30.) Two years after operation. Sixty degrees of motion (Gill, A B J Bone & Joint Surg 17 1, 1935)

ner, a new acetabulum may be constructed here by making as deep an excavation on the side of the ilium as is possible without penetrating the inner table A bone flap is turned down over the head, and the space above it is filled with small wedges and firmly packed chips of bone. If constant abduction is maintained until the cast has been applied, the head of the femur will not slip out of the excavation and displace the bone shelf. Adductor tenotomy may be necessary to secure sufficient abduction.

If these operations are done properly a sufficient and stable acetabulum can be formed above the primary socket Lordosis has been largely corrected by bringing the head forward. Stability is assured. Motion in flexion will vary between 30 and 90 degrees, depending, I believe, largely on the amount and the nature of the deformity of the head of the femur, but always sufficient to permit the patient to sit comfortably.

3. Osteotomies of the Femur

Reconstruction of the hip may not be advisable in old high dislocations in which the head of the femur lies too high or when it cannot be brought forward to a position above the primary acetabulum, and also in bilateral dislocations when the danger of too much limitation of motion in both hips cannot be risked. However, the reconstruction affords more stability on weight bearing than does the osteotomy.

Osteotomies, devised by Lorenz, Schanz, and others, are designed to alter the direction of the thrust of the lower extremity against the pelvis by abduction of the femur below the site of the osteotomy, or by a combination of abduction and displacement of the lower fragment inward against the side of the pelvis. If, after union of the fragments has taken place, the abducted extremity is brought inward to the longitudinal line of the body, the pelvis will be tilted downward on the affected side, provided the upper fragment is secured to the side of the pelvis. Thus the angle of incidence of the thrust of the body weight against the side of the pelvis is lessened, i.e., it is directed more perpendicularly against the pelvis, with a resulting increase in stability (Figs. 32 and 33). However, if the upper fragment is



Fig. 32 S R., female, adult Bilateral dislocation

not fastened securely to the side of the pelvis, its superior portion will move outward and downward and the inferior portion upward and inward as the leg is adducted. This rotary mo-



Fig 33 S R (Same as Fig 32) Osteotomy with abduction Note tile of pelvis upward on the opposite side

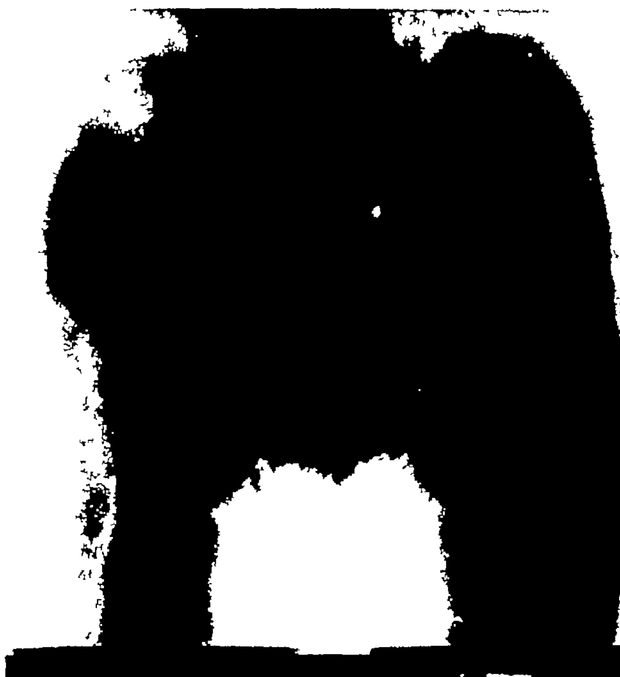


Fig. 34 E W , female Result of osteotomy with abduction Note that pelvis is not tilted up on opposite side materially

tion is due to the fact that the capsule is attached to the base of the neck between the two ends of the fragment and acts as a point of support on which the fragment rotates The upper end of the lower fragment is carried in-

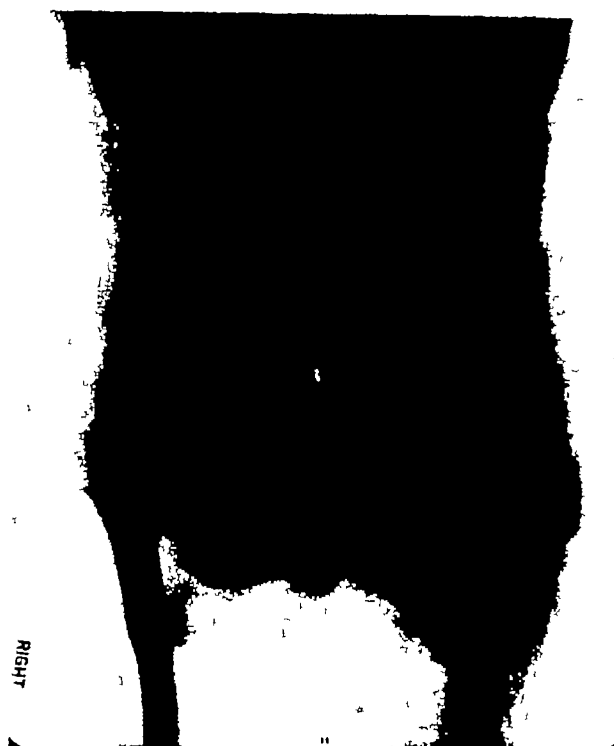


Fig 35 G C , male 5 16 45 Old bilateral dislocation



Fig 36 G C (Same case as Fig 35) 9 17 45 Lorenz type of osteotomy

ward more closely against the side of the pelvis and thus secures a better thrust against it, but the pelvis is not tilted by adduction of the

leg to the mid-position (Fig 34).

In the Lorenz bifurcation the lower fragment is abducted and is also thrust inward against the pelvis and into the primary acetabulum (if there is one), or beneath some ledge on the side of the pelvis. Unfortunately, in older cases the side of the pelvis may be as smooth as a wall. In such case the chief factor for increased stability is the abduction of the femur (Figs. 35 and 36).

REFERENCES

Gill, A Bruce Operation for Old or Irreducible Congenital Dislocation of the Hip J Bone & Joint Surg 10 696-707 (Oct , 1928)

Gill, A. Bruce, Orr, Theodore E , and Jepson, Paul N Congenital Dislocation of the Hip Stretched by Means of a Turnbuckle Cast. J Bone & Joint Surg 16 905-908 (Oct , 1934)

Gill, A. Bruce Plastic Construction of an Acetabulum in Congenital Dislocation of the Hip - The Shelf Operation J Bone & Joint Surg 17 48-59, (Jan , 1935)

Gill, A Bruce An Evaluation of Present-Day Methods of Dealing with Congenital Dislocation of the Hip J Bone & Joint Surg 18 487-509 (April, 1936)

Gill, A Bruce End Results of Bloodless Reduction of Congenital Dislocation of the Hip J Bone & Surg 25 1-40 (Jan , 1943)

Gill, A Bruce Congenital Dislocation of the Hip Nelson's Loose-Leaf Surgery, 1946

Course No. 8

THE ELBOW JOINT

Lecturers

Harold B. Boyd, M.D., Surgical Approaches to the Elbow Joint

T. L. Waring, M.D , Surgical Lesions of the Elbow

SURGICAL APPROACHES TO THE ELBOW JOINT

Harold B Boyd, M D.

IN THIS PAPER are described the commonly used surgical exposures of the elbow joint which have been found of most practical value. No attempt is made to catalog all the described exposures.

Posterior Approach

For extensive exposure of the elbow joint, the posterior approach is the easiest and safest. The incision is begun on the posterior aspect of the arm approximately a hand's breadth* above the elbow and, from this point, is carried downward to the posterolateral aspect of the olecranon (Fig. 1). The skin and the superficial and deep fascia are divided in the line of the incision. The margin of the wound is retracted, exposing the tendon of the triceps. A triangular flap of triceps tendon is outlined by sharp dissection, the base of the triangle comprising the insertion of the triceps into the olecranon, the apex in the upper portion of the incision In the upper two-thirds of the fascial flap, muscle tissue is encountered beneath the triceps tendon, whereas, in the lower third of the triangular flap there is no muscle beneath the triceps tendon. In dissecting this flap downward, the fascial portion of the triceps tendon is separated from the muscle by sharp dissection, if left attached, the muscle will be deprived of its blood supply and will atrophy. In the upper portion, therefore, this tendinous flap is thin, while the lower portion includes the entire thickness of the triceps tendon

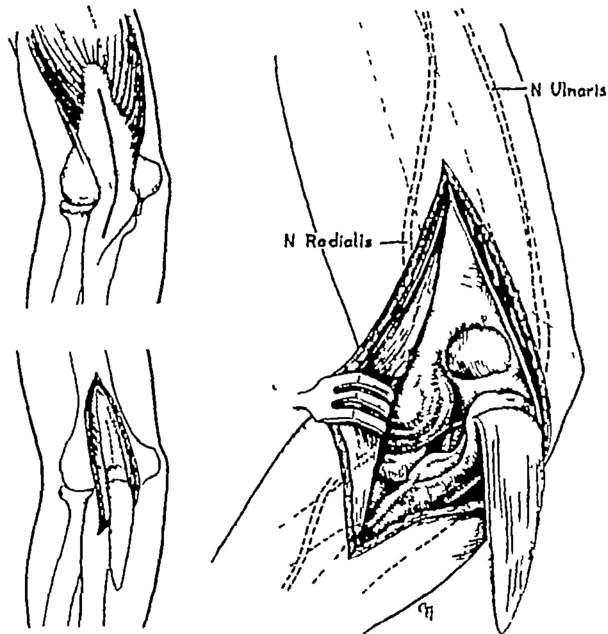


Fig 1 Posterior approach

The fascial flap is retracted distally and the underlying triceps muscle is divided per-

*In dealing with hand and finger breadths, these measurements should be interpreted in terms of the patient s hand and fingers, and not the surgeon's

pendicularly in the midline down to the bone, taking care not to injure the radial nerve in the upper angle of the incision. The lower end of the humerus, including the olecranon fossa, may now be exposed by subperiosteal stripping of the tissues, either medially or laterally, as desired. If the exposure is to be carried medially past the ulnar groove, the ulnar nerve should be exposed in the groove, freed well above and below the joint, and gently retracted before subperiosteal stripping of the area is completed. Further exposure of the elbow joint is obtained by subperiosteal dissection of the anconeus muscle from the lateral surface of the upper portion of the ulna. Lateral retraction of this muscle will expose the head of the radius, the posterior aspect of the humeroradial joint, and the lateral aspect of the humeroulnar (Fig. 1). Thereafter, either flexion or extension of the elbow will bring into view the major portion of the posterior and inferior aspects of the joint.

In closing the incision, the divided fibers of the triceps muscle are sutured together and the flap of the triceps tendon is replaced and sutured into position. The deep fascia, superficial fascia, and skin are closed in the routine manner.

This approach is useful for open reduction of comminuted fractures of the lower end of the humerus, old posterior dislocations of the elbow, and resection, fusion, or arthroplasty of the elbow.

Anterior Approach

Henry[1] describes an extensive anterior approach to the elbow joint, which he states is a variation of Fiolle and Delmas' exposure of the vessels and nerves at the front of the elbow. The incision extends along the lateral border of the biceps in the lower portion of the arm, curves medially to the center of the antecubital fossa, thence down the center of the anterior surface of the forearm for approximately a hand's breadth below the elbow.

The skin is retracted, exposing the cephalic vein, the biceps tendon and the lacertus fibrosus. The vein is ligated and the lacertus fibrosus is divided, together with the deep fascia of the forearm. In dividing the lacertus fibrosus, care must be taken not to injure the median nerve or the brachial artery, which lie beneath it. The lower portion of the brachialis muscle is divided and retracted, exposing the lower end of the humerus, as in Henry's anterolateral approach to the humerus.

The interval between the brachioradialis and extensor muscles on the lateral side, and the pronator teres and the flexor muscles on the medial side is developed by blunt dissection. The brachial artery gives off its terminal branches, the radial and ulnar arteries, near the medial aspect of the lower portion of the biceps tendon. To facilitate retraction and exposure of the anterior portion of the elbow joint, the stem of the radial recurrent artery is ligated close to the radial artery. The radial recurrent arteries bind the radial artery to the lateral muscles and prevent retraction of the space between the extensor and flexor groups of muscles. After ligation of the arterial stem, the forearm is pronated and the muscle groups retracted, this gives good exposure of the structures of the antecubital fossa and the anterior ligaments of the elbow joint. Division of these ligaments reveals the anterior portion of the joint.

This is an excellent approach to the vessels and nerves on the anterior aspect of the elbow, but is rarely needed for exposure of the joint, other, less formidable and hazardous incisions can usually be employed to expose the joint. In exposing the joint through this incision, extreme care must be used in retracting the vessels, as trauma due to retraction may cause a segmental spasm of the artery, and consequent impairment of circulation of the forearm. In the event this occurs, elevation of the extremity, active motion of the fingers, and sympathetic bloc will be of value.

Lateral Approach

The incision begins three to four fingers' breadth above the lateral epicondyle of the humerus, extends down the lateral surface of the arm to the epicondyle, thence down the posterolateral surface of the forearm a distance of two to three fingers' breadth. The tip of the lateral epicondyle is exposed. The lateral border of the humerus is exposed from below upward by developing the interval between the origin of the brachioradialis and extensor carpi radialis longus anteriorly, and the triceps posteriorly. In the upper angle of the incision, one must avoid damage to the radial nerve as it enters the interval between the

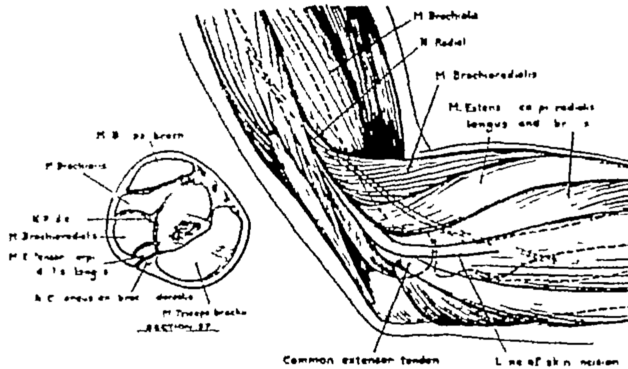


Fig 2 Lateral approach

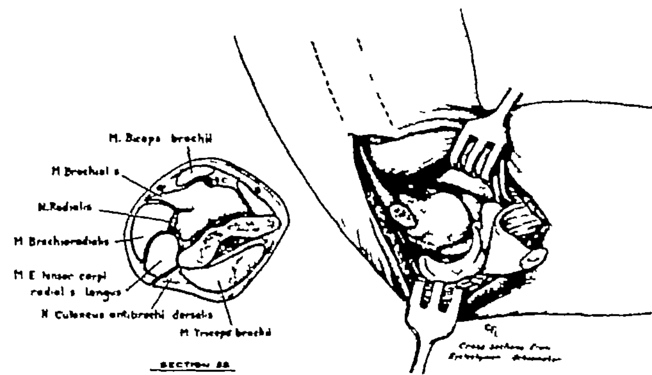


Fig 3 Lateral approach completed

brachialis and brachioradialis muscles (Fig 2) The common origin of the extensor muscles from the lateral epicondyle may be dealt with in one of three ways The common origin may be separated from the humerus together with a thin fleck of bone, using a small osteotome, or it may be divided just distal to the medial epicondyle In any event, the common origin is displaced distally, revealing the radiohumeral joint. One should be careful not to damage the deep branch of the radial nerve as it enters the two planes of the supinator muscle By the third method, the interval between the extensor muscles and the anconeus muscle is developed, as in the exposure of the head of the radius (to be described later). The origins of these muscles are stripped subperiosteally and the muscles retracted anteriorly revealing the radiohumeral joint.

The origins of the brachioradialis and extensor carpi radialis longus are elevated subperiosteally and the capsule of the joint is divided, exposing the lateral aspect of the elbow joint (Fig 3) In closing the wound, the origin of the extensor muscles is reattached. The wound is closed in the routine manner

In operating for fracture of the external condyle of the humerus, this is an ideal approach. As the common extensor origin is attached to the condylar fragment, it is unnecessary and inadvisable to disturb the origin of the extensor muscles

Exposure of the Head of the Radius

An oblique incision is begun over the posterior surface of the lateral epicondyle of the humerus and extended distally and medially to a point over the subcutaneous border of the ulna, three fingers' breadth below the tip of the olecranon The deep fascia is divided in the line of the incision, and the fascial plane between the anconeus and extensor carpi ulnaris muscles is developed Retraction of these muscles reveals the joint capsule of the radiohumeral joint and the transverse fibers of the upper portion of the supinator muscle. The joint capsule is then incised, exposing the head of the radius. By retracting the supinator fibers downward and anteriorly, the neck of the radius may also be brought into view (Fig 4).

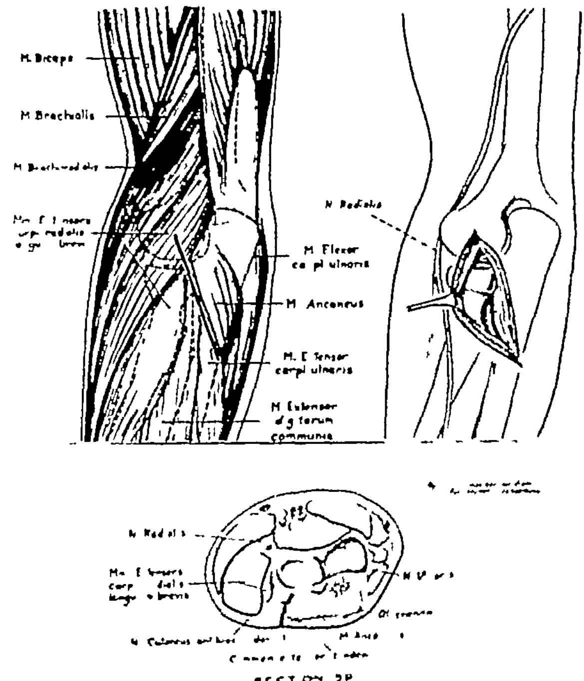


Fig 4. Exposure of the head of the radius

The deep branch of the radial nerve is protected between the two planes of the supinator muscle.

Medial Approach

The incision described by Campbell[2] is made over the tip of the medial epicondyle of the humerus, extending from approximately two and one-half fingers' breadth above the joint to three fingers' breadth below. The subcutaneous portion of the medial epicondyle is exposed. The medial supracondylar ridge of the humerus is then exposed from below upward along the medial intramuscular septum, between the brachialis muscle anteriorly and the triceps muscle posteriorly. The ulnar nerve is found in the ulnar groove at the back of the epicondyle. The nerve should be gently freed for three centimeters above and below the condyle, and retracted with a moist soft rubber drain loop (Fig. 5).

After securing protection for the ulnar nerve, the tip of the medial epicondyle is divided with an osteotome, the epicondyle with its muscular attachments is displaced distally, and the capsule of the joint incised. A valgus strain is placed upon the elbow producing a subluxation, this affords exposure of the medial two-thirds of the joint (Fig. 6). In closing the wound, the medial epicondyle may be held in position by a small rustless steel nail, the head of the nail being left so that it is palpable beneath the skin. The remainder of the wound is closed in the routine manner. The nail should be removed after six weeks. This may be done with local anesthesia, through a short incision.

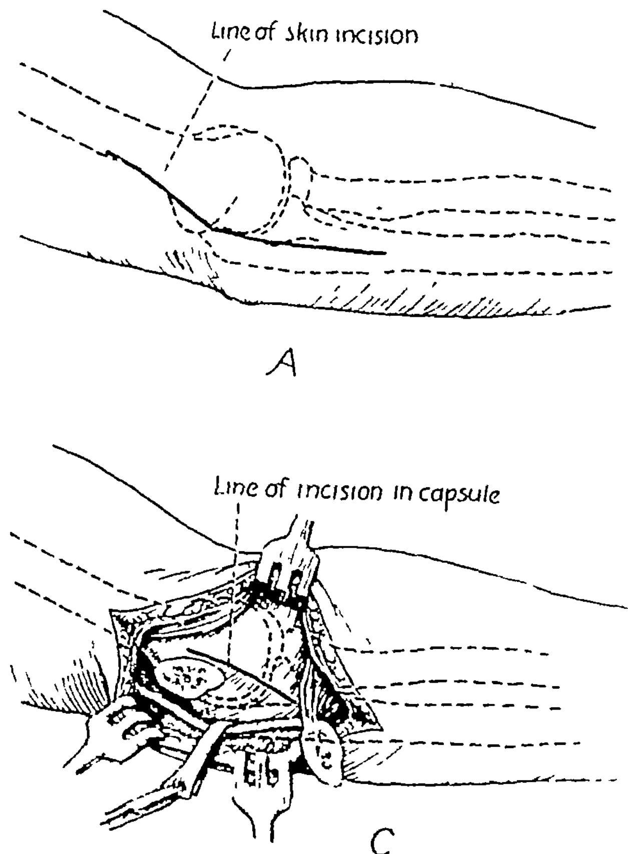


Fig 5 Medial approach

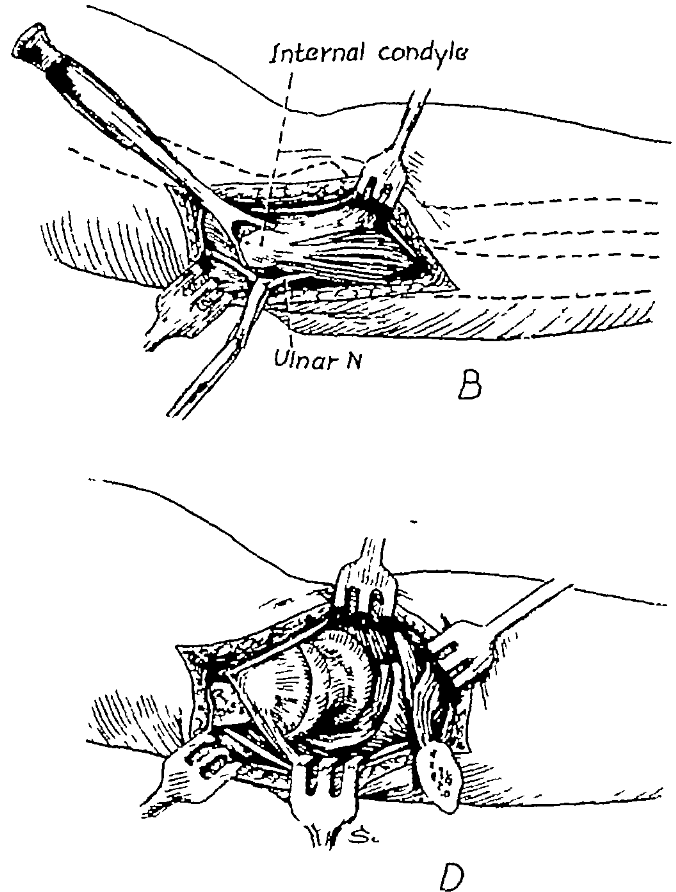


Fig 6 Medial approach completed

Exposure of the Upper Third of the Ulna and the Head and Neck of the Radius through One Incision[3]

The incision begins two fingers' breadth above the tip of the olecranon at the lateral margin of the triceps tendon, and extends

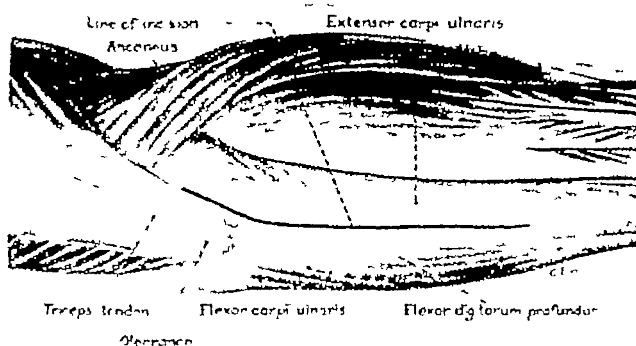


Fig 7. Skin incision for exposure of the upper third of the ulna and the head and neck of the radius. (Reproduced by permission from Surg, Gynec. & Obst 71 86.)

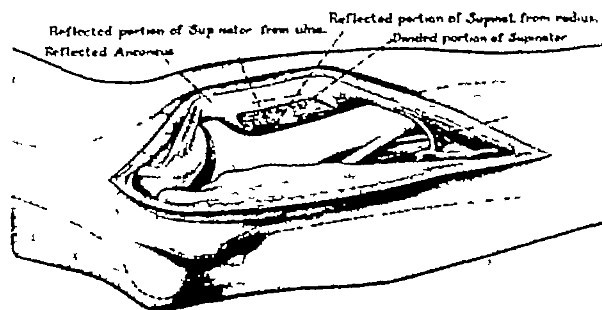


Fig 8 Exposure of the upper third of the ulna and head and neck of the radius (Reproduced by permission from Surg, Gynec & Obst 71 86)

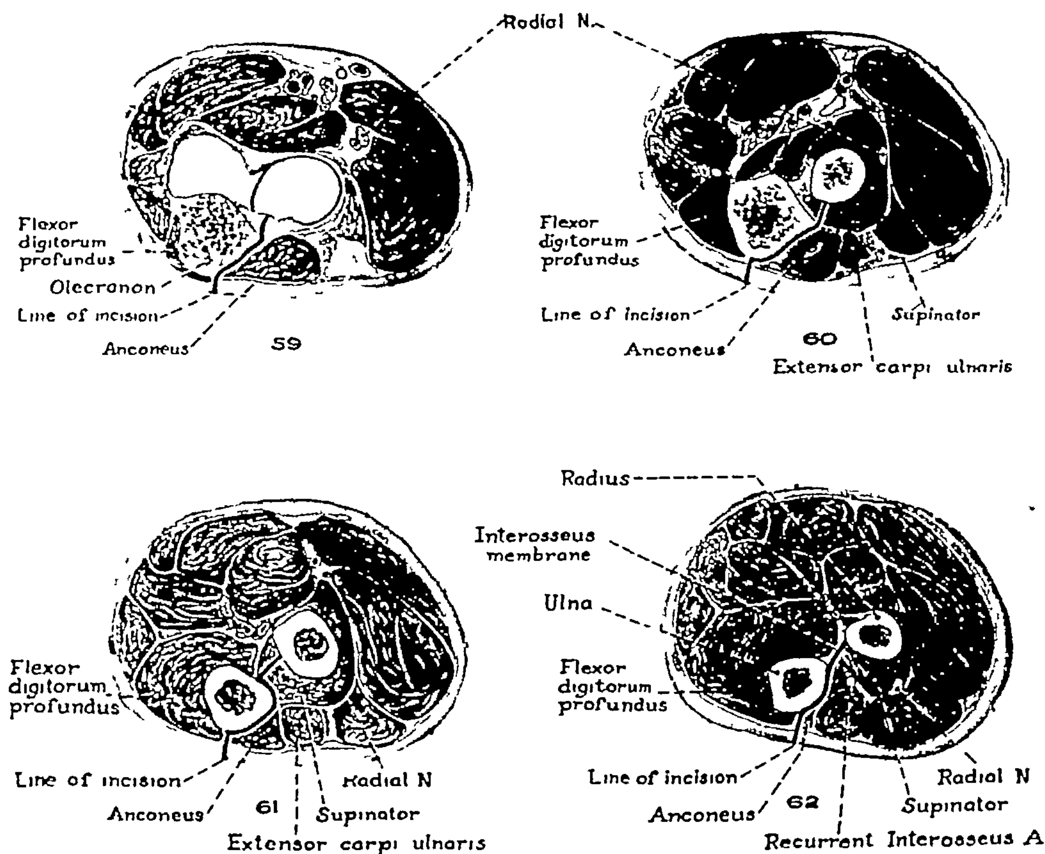


Fig 9. Cross sections showing the relation of the exposure to the radial nerve at the level of the elbow joint (59) and approximately one, two, and three fingers' breadth below the elbow joint (60, 61, and 62). (Reproduced by permission from Surg, Gynec & Obst 71 88)

downward along the posterior border of the ulna for a sufficient distance to permit the desired exposure of this bone (Fig. 7). Dissection is continued down to the ulna along the intermuscular septum between the anconeus and extensor carpi ulnaris on the radial side and the flexor digitorum profundus on the ulnar side. The attachments of the anconeus and extensor muscles are elevated from the ulna subperiosteally, exposing the lateral surface of the shaft of the bone. The origin of the supinator muscle is elevated at its ulnar attachment. The capsule of the elbow joint and the annular ligament are divided close to their attachments to the upper portion of the ulna. The supinator muscle, the capsule of the joint, and the annular ligament are then reflected laterally, exposing the head and neck of the radius (Fig. 8).

The deep branch of the radial nerve lies between the two planes of the supinator muscle and is thus protected from injury. (See cross sections redrawn from Eycleshymer and Shoemaker, Fig. 9.) The first is at the level of the elbow joint, the last about a hand's breadth below.

This is a convenient exposure for fractures of the ulna associated with dislocation or fracture dislocation of the head of the radius.

REFERENCES

1 Henry, Arnold K Extensile Exposure Applied to Limb Surgery, Edinburgh, E and S Livingstone, Ltd , 1945
2 Campbell, Willis C Incision for Exposure of the Elbow Joint, Am J Surg 15 65 (1932)
3 Boyd, H B Surgical Exposure of the Ulna and Proximal Third of the Radius Through One Incision Surg , Gynec & Obst 71 86 (July, 1940).

SURGICAL LESIONS OF THE ELBOW

T. L. Waring, M.D.

The conditions which affect the elbow are not peculiar to it alone, but are found in the other joints as well. In this discussion we will confine ourselves to this particular region, as it is the subject of the course The conditions to be discussed are as follows congenital and developmental anomalies, neoplastic disease (osteochondromatosis), tuberculosis, certain traumatic manifestations, exclusive of fractures, osteochondritis dissecans, and juvenile osteochondritis.

Congenital Anomalies--The chief deformities met with are due to fusion of the epiphyses producing bony ankylosis, such as radio-humeral and ulna-humeral synostosis, fusion of the epiphyses or diaphyses of the upper end of the radius and ulna such as radio-ulnar synostoses, subluxation and dislocations of the head of the radius and patella cubiti.

Radio-Humeral and Ulna-Humeral Synostosis--This condition is very much rarer than radio-ulnar synostosis. According to H. S. Murphy and C. G. Hanson[1] only 24 cases of radio-humeral synostoses have been reported in the literature to 1945. Steindler[2] reports a case of ectrodactyly which showed a complete synostosis of the ulna and the humerus in the right. In the x-ray the ulna was united with the humerus at almost right angle, whereas the radius was united with the humerus on the other side in extended position. Murphy and Hanson[1] report a case of bilateral congenital humeroradial synostoses. Steindler[2] states that early arthroplasty or resection of a portion of the elbow joint is indicated. However, he does not say that he has done this. Campbell[3] in his "*Operative Orthopedics*" does not mention this condition. Mouchet and Saint-Pierre[4] report an attempt at surgery on a bilateral case of humeral radial synostosis. The ankylosis was sectioned, the extremities shaped to form a joint, and a musculo-aponeurotic flap was inserted to prevent ankylosis. They did not give the end result study on their case.

Radio-Ulnar Synostoses--This is a much more frequent condition than the above. In this condition there is a bony bridge between the proximal ends of the radius and ulna, which completely inhibits any pronation or supination. Rarely it may involve both the proximal and distal ends. The treatment is operative, resection of the synostosis and the interposition of soft tissue to prevent recurrence of the bony bridge. The results are very discouraging. Steindler[2] reports four cases with the following end results, good 0, fair 1, poor 3 His standard good, pro-and-supination range of 90 degrees or more, fair, 60-90 degrees

Congenital Dislocation of the Head of the Radius--This is a relatively rare condition. It may be primary, or secondary to a total or partial defect in the ulna. For the simple primary dislocation resection of the head of the radius is indicated, for the secondary dislocation the correction of the ulnar deformity followed by resection of the radial head.

Patella Cubiti. In this condition radiographs show a large sesamoid bone occupying a position on the dorsum of the elbow which simulates the appearance of the patella of the knee joint. According to Habbe,[5] the condition was probably first described by Dr. F. J. Cotton of Boston in 1900 He did not use the above term, the title of his paper being, "Separation of the Epiphysis of the Olecranon." Keinboch, in 1903, was the first to apply the term "patella cubiti" to this condition. It has since been described by a number of authors. The latest treatise on this subject is that by J. E. Habbe of Milwaukee, Wisconsin, in the October 1942 issue of the American Journal of Roentgenology and Radium Therapy. He gave a report of four cases with excellent illustrations As to the etiology of this condition, there appears to be considerable divergence of opinion among the various authors. The opinion is divided between the theory of traumatic origin and the theory of congenital anomaly. Habbe suggests a third possible explanation, that of epiphysitis of the olecranon. In Habbe's four cases it was possible to show in three of them an etiological relationship between childhood trauma and the formation of the patella. In one of his cases the past history was absolutely negative for trauma He says "In true patella cubiti any trauma factor significant to the development of the condition must be one occurring in childhood or adolescence." No operative treatment is indicated, if there has been trauma to the elbow, immobilization is indicated until the reaction has subsided A medical-legal angle is usually present in these cases.

Persistent Olecranon Epiphyses in Adults--This condition is often confused with patella cubiti, the latter is generally conceded to be of embryonic origin, although this is not a settled question It is a completely isolated accessory bone, articulating and mobile, while the former is a simple persisting epiphysis, usually found bilaterally and having a familial tendency. O'Donaghue and Sell[6] reported the first case in American and English literature. Their case showed this condition to be bilateral, in addition there was present bilateral tripartite patellae. Recently the author encountered a case which he believes falls into this category. The case history is as follows

R S P Male. Age 25 Entered the hospital on 4-27-46 because of a painful swollen right elbow. He stated that on 4-26-46 at 8 30 p.m. while skating he fell on the point of his elbow. X-rays showed what appeared to be a fracture of the olecranon process of the ulna. Examination of the right elbow showed diffuse swelling and limitation of motion. Operation was performed on the day of admission. The fragments of the olecranon were smooth and apparently covered by fibrous cartilage. A tear in the triceps tendon was the only fresh trauma found. The fibrous cartilage was removed from the fragments, the fragments were approximated and held in position by a wire loop and one vitallium screw. The triceps aponeurosis was repaired Since the operative findings were other than suspected, the patient was questioned as to preceding trauma. He admitted that one year previous to the last injury he had injured his elbow but that it was not of great enough significance to require medical care X-rays taken on 5-23-47 showed that the fragments were maintained in position but no union was present. A recent communication from the patient stated that he had good function in his elbow (Figs. 1 and 2).

Neoplastic Conditions of the Elbow - Osteochondromatosis--The condition of osteochondromatosis is a rare pathological process causing the formation of synovial osteochondroma in tendon sheaths, bursae, and joints. They are not associated with osteoarthritis or



Fig. 1 R S. P. Preoperative x-rays taken at time of injury.



Fig. 2 R. S. P. X-rays taken one month postoperative, showing maintenance of position, but no bony union. A recent communication from the patient stated that he had good function in his elbow.

synovitis. The synovial membrane in this condition forms bodies which contain cartilage or bone, or both bone and cartilage. At first these bodies are attached by means of synovial membrane pedicles to the synovial membrane. These pedicles easily become detached, and allow the bodies to float free in the joint cavity.

The etiology of this condition is not definitely known. Henderson and Jones[7] consider them to be benign neoplasms and present considerable evidence to substantiate their theory. Lannec, in 1813, was the first to describe this process, and since this time much interest has been shown in this condition. Hugh T. Jones[8] has written a very excellent treatise on the subject. He believes that the origin of these bodies is in the stratum synoviale of the synovial membrane and that they may start as either chondromas or osteochondromas, that the cartilage in the undetached bodies may be purely hyaline, fibrous, calcified, or a combination of these forms, and the bone growth in the undetached bodies is developed directly from connective tissue by the membrane method and also by preformation in cartilage. The form most frequently found in his cases was that of a spherical shell of bone surrounded by cartilage and fibrous tissue and filled with vascular tissue and spurs of bone. In all the detached bodies the bone was necrotic.

There are a number of theories as to the etiology. These are the infectious, traumatic, embryonic, and the neoplastic. There is very little evidence that infection plays a major role. Trauma is given an important place in the recorded histories in the literature and probably plays some part in the etiology of this condition. Embryologic - All the tissues involved in this process are developed by differentiation of the same mesenchymal tissue and it is quite logical to assume that it plays

a definite part. Neoplastic - The various stages of differentiation found in the pathologic material suggest the neoplastic theory. The treatment is surgical removal of the loose bodies and a synovectomy if possible. The following case history is of considerable interest in that it presents several points of interest as to the etiology (Figs. 3 and 4).

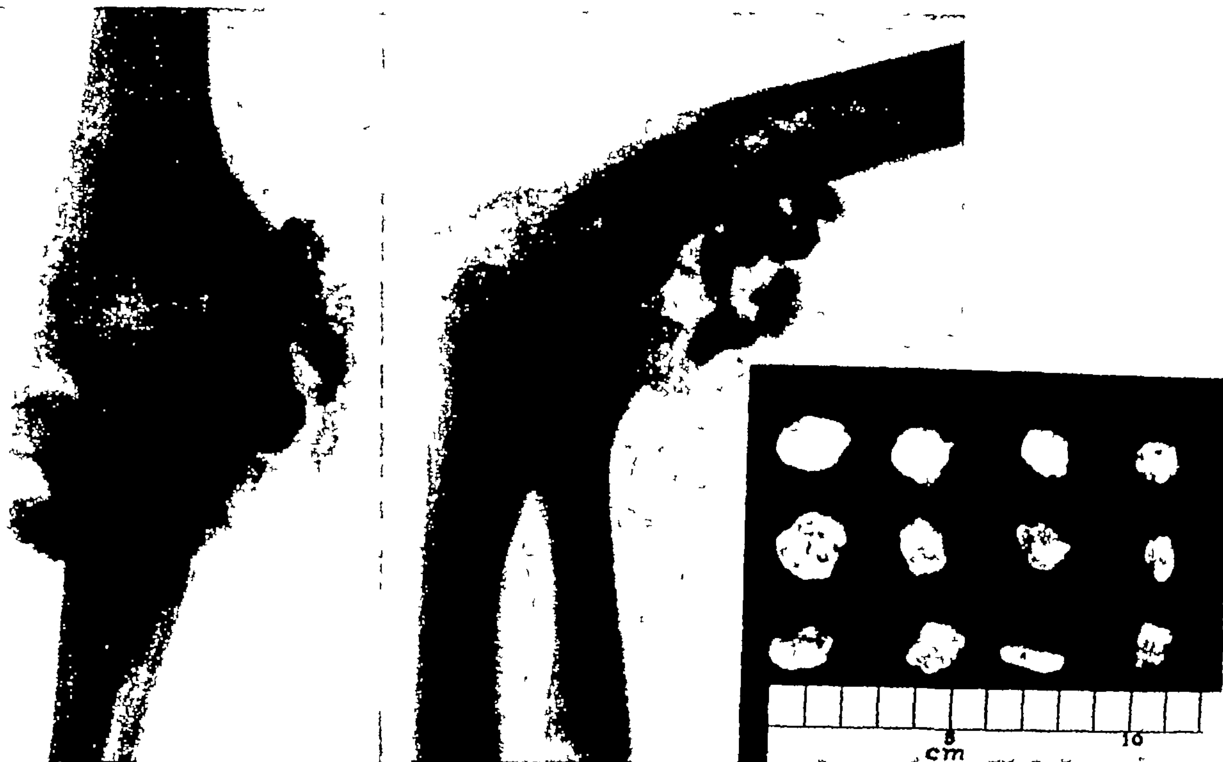


Fig. 3 & 4 C. H Preoperative x-rays showing the free bodies in the olecranon fossa and in region of internal epicondyle.

A.M. Age 37. Was admitted to the hospital on 10-1-46 because of a swollen painful right elbow He gave the following history 15 years ago while wrestling he was felled, hurting his right elbow. He recovered fully from this injury Since the original injury he had three additional injuries to his elbow, each succeeding injury causing more and more disability. He complained of pain, swelling and inability to use his elbow. He stated that since the last injury, in June 1946, he has been unable to use it, due to severe pain and swelling. In the past 4 - 6 weeks he had lost 25 pounds in weight and had been unable to sleep because of pain. Examination of the right elbow revealed it to be swollen and tender. It was held in a position of 100 degrees with only a few degrees of motion from this position. A mass was palpable over the medial surface of the elbow. There was a mild increase in local heat but no redness or induration Temperature 101 degrees. X-rays of the elbow showed multiple loose bodies scattered throughout the anterior compartment of the elbow joint. There were also noted some changes in the region of the radiohumeral articulation. Due to the elevated body temperature and the appearance of the elbow it was felt that there was a secondary infection superimposed on his original condition. He was, therefore, treated conservatively for 15 days with hot packs and penicillin. On 10-15-46 the right elbow joint was approached through both a medial and a lateral incision. When the joint was opened the synovia was found to be thickened and hypertrophied with villous formation, the synovial fluid was unusually thick and cloudy. A culture was taken of this material because it was thought to be purulent. Numerous osteochondromata were removed (Fig. 5). Some of the smaller were still at-



Fig 5 C. H. Preoperative x-rays showing the free bodies in the olecranon fossa and in region of internal epicondyle.

tached to synovial membrane by fibrous pedicles. The joint showed evidence of chronic irritation. The culture was negative. Histopathology, osteochondromata. After operation the temperature subsided, wound healed by primary intention. No follow-up examination has been obtained as the patient did not return as requested.

Inflammatory Conditions of the Elbow - Tuberculosis of the Elbow--Of all the joints of the upper extremity, the elbow is probably the one most frequently invaded by tuberculosis, however, when compared with tuberculosis in the lower extremity, it is a fairly rare condition. Girdlestone[9] reports only 19 cases treated at the Wingfield-Morris Orthopaedic Hospital in a period of fifteen years. Cleveland[10] reports 10 cases of tuberculosis of the elbow in 500 cases of bone and joint tuberculosis. Steindler reports 19 cases in 500 cases of tuberculosis of the bones and joints. Reviewing the cases at the Campbell Clinic we find only 22 cases.

In contrast to tuberculosis of the lower extremity, tuberculosis of the upper extremity including the elbow favors decidedly the period of adult life. Before discussing the treatment it will serve us well to discuss the question of the positive diagnosis of tuberculosis, as upon this depends the soundness of our treatment.

Perusal of the literature, especially the older literature on this subject, makes one doubt at times the correctness of the diagnosis, especially upon close scrutiny of the end results in the conservative treatment of joint tuberculosis. The correctness of the diagnosis is also being more seriously challenged since the development of the operative methods for treatment of this condition. The later reports show a rather large percentage of inaccurate diagnoses.

Allan DeForest Smith[11] reports a high percentage of errors in diagnosis, of 39 cases of proven joint tuberculosis an incorrect diagnosis was made in 14 on admission (32 months was the average duration before admission) a diagnostic error of 38%. Milgram[12] reports an error in diagnosis of 38.7% in 142 cases.

Yet Rollier[13] reports 85% cures by conservative treatment.

The most reliable means of making a positive diagnosis is by surgical biopsy. By this almost 100% of positive diagnoses can be made This procedure is practical in all joints that are accessible to aspiration or biopsy. Cleveland[10] made a positive diagnosis in 121 of 157 available joints by the study of tissue or exudate. He also proved it in 16 of 173 spines. The elbow is certainly one of the most accessible of all joints for this procedure. There is considerable controversy regarding biopsy for diagnosis Many feel that since the percentage of clinical diagnosis is low, and the error is great, that exploratory operation should be done as a routine procedure. Others feel that biopsy should be performed only in cases in which surgical intervention is indicated as soon as the diagnosis is established.

In considering the operative treatment for tuberculosis regardless of the joint involved, as Steindler[2] so aptly states, "we must be certain that the tuberculous disease is in a stage in which operation will be beneficial." Cleveland[10] says "It is absolutely necessary to consider patients with joint tuberculosis from the standpoint of their general infection with the disease."

Having fulfilled the above criteria, we are faced with the problem of what surgical procedure will fulfill our requirements. On this point there is considerable divergence of opinion. Campbell[3] states "In adults, excision is the treatment of choice, especially when destruction is excessive." He further states "This method will give a serviceable though rather unstable member in the majority of cases." Steindler[2] reserves resection for severe cases, particularly the fungus type with destruction in the presence of sinuses and secondary mixed infection, and for older individuals in whom it is felt a successful fusion will not be accomplished because of their lack of resistance. Cleveland[10] says "While a successful fusion of the elbow joint will arrest the disease, it is followed by more disability than in any other joint. For this reason we have been doing resections of this joint in favorable cases We cannot as yet draw any conclusions on these few cases." Steindler[2] favors an intra- and extra-articular fusion before there is extreme destruction of bone constituents. He states "It is the only operative procedure (with the exception of very rare intra-osseous excision) which offers a reasonable assurance of permanent cure." Girdlestone[9] advises in most cases excision and arthrodesis. DeForest Smith prefers arthrodesis in spite of the fact that motion is sacrificed, because it is much more certain to effect a cure and because the arm can be used for heavy work without fear of recurrence.

Arthroplasty is not recommended by most authorities, and the majority of them state that it is absolutely contra-indicated in tuberculosis. It would seem that the consensus of opinion is that fusion offers the best chance for cure of tuberculosis of the elbow. The operative treatment is rarely indicated in tuberculosis of the elbow in children. A conservative course is considered preferable because in children the regenerability is much greater than in adults and even osseous cases with considerable destruction may end with full mobility. This statement is refuted by a number of authorities DeForest Smith says "There is no proof that children respond more favorably than others to so-called conservative treatment and, in fact, reliable records show that they do not. The results of fusion operations in young children on the contrary have been better even than adults." Occasionally amputation is indicated in cases which have progressed beyond the reach of the lesser surgical procedures.

Traumatic Conditions of the Elbow - Pitchers Elbow (Traumatic Arthritis)--This condition may occur in any elbow which is subjected to unusual stress and strain, especially so in the elbows of baseball pitchers. It may occur at any time during their career but usually requires a number of years to develop. The condition is characterized by the formation of loose bodies in the joint which limit the motion They are usually found in the region of the olecranon fossa, the internal epicondyle or at the tip of the coronoid process. An ulnar neuritis is very frequently present when they occur in the region of the internal epicondyle. This neuritis is caused by an irritation of the nerve owing to the free body or bodies. Bennett[14] feels that the condition falls within the group of the so-called osteochondritis and that the bodies are developed by trauma to the hyaline cartilage, causing hypoplasia of this structure. A fairly typical case history is as follows

C.H., pitcher for New York Giants, age 35, was admitted to the hospital on 8-8-38. He stated that 3 years previously he noticed that he was unable to fully extend his elbow and that the deformity was gradually increasing. He had no particular pain, but did notice much stiffness and stated that after he had finished a game his elbow was exceedingly sore and moderately swollen. His "warm up" periods became progressively lengthened. During the 1938 season he had frequent lockings when sudden efforts at extension were made. These "lockups" were very painful. Physical examination showed the following Left upper extremity well developed, moderate swelling of forearm centered around the medial humeral condyle. Motion in elbow was from 160-60 degrees. There was tenderness over the course of the ulnar nerve, no sensory or motor disturbance, supination limited 5 degrees by pain. X-ray examination revealed three loose bodies present in the region of the medial epicondyle and what appeared to be one loose body in the olecranon fossa. There was also noted some reaction about the lateral epicondyle. At operation on 8-22-38 through a dorso-lateral incision the olecranon process was exposed, the elbow joint was then entered both medially and laterally through longitudinal incisions on both sides of the olecranon process. The joint capsule and synovial membrane were found to be thickened. The loose bodies were removed. It was found that the articular cartilage was roughened throughout the joint. At follow-up examination on 4-3-39, the following note was made Movement in the elbow is free and painless. No grating felt. Motion is from 160-40 degrees, pronation and supination normal. Patient stated that he was able to pitch without any return of the former disability, however, at this return visit he complained of pain in the left shoulder. This responded to physical therapy.

Bennett[14] in his article entitled, "Lesions of Professional Baseball Pitchers," also describes a shoulder lesion. For details you are referred to his original article.

Conditions in the Elbow of Undetermined Etiology.- Osteochondritis of the Elbow--This condition may occur in several different locations in the components that make up the elbow joint, the most common site being the capitellum of the humerus, a less frequent site, the internal condyle.

Osteochondritis Dissecans of the Supratrochlear Septum--This is a relatively rare condition which was first described by W. E. Crysler and H. S. Morton.[15] In their review of the older literature on loose bodies in the supratrochlear fossa, they are inclined to believe, though they do not definitely state so, that a number of the cases reported are due to the above condition, namely, osteochondritis dissecans. The majority of the cases reported have been called sesamum cubite. Morton and Crysler[15] presented six cases of this condition in which the histological findings were those of osteochondritis dissecans. According to Hirsch,[16] the septum is always present until the age of 7 years, after which the bony plate occasionally becomes absorbed to form the supratrochlear foramen. Hirsch quotes examinations by Martin which revealed the presence of the supratrochlear foramen in 58% of Arkansas Indians studied, and in 21.7% of African Negroes, but showed the proportion to be only 4.2% in white Americans. The supratrochlear septum when present consists of thin transparent layers of bone. In the motion of the elbow, the olecranon fossa receives the summit of the olecranon in extension, the coronoid fossa receives the coronoid process of the ulna in flexion. Morton and Crysler state. "In the extremes of movement of this joint, there is apt to be repeated trauma to the septum, which may lead to osteochondritis dissecans." The treatment for this condition is the operative removal of the free body.

Osteochondritis Dissecans of the Elbow--Osteochondritis dissecans is an aseptic necrosis of a segment of subchondral bone arising from the articular surface of the ends of the bones of the extremities, resulting almost always in an osseous cartilaginous free body into the joint. They usually occur singly, but may be multiple, and rarely number more than 2 or 3. The medial femoral condyle of the femur is most often affected, the capitellum of the humerus running close second. It is more frequently seen in males, during either late adolescence or early adult life. The condition may be unilateral or bilateral, approximately 20% of the cases are found to be bilateral. The etiology of the condition remains rather obscure. The main theories expounded are. trauma, vascular disturbances, constitutional and familial factors, fat and bacterial embolism, local osteoarthritis, etc. Phemister[17] in his

most excellent treatise entitled, "The Causes of and Changes in Loose Bodies Arising from the Articular Surface of the Joint" states "From a consideration of the evidence for and against the various theories of causation of the classical loose bodies that have here been enumerated, we are forced to the conclusion that an entirely satisfactory explanation has not yet been offered " Symptomatology - There are usually no characteristic symptoms. The patient usually gives a history of a vague or indefinite trauma, at other times the story of trauma is quite definite. At first the symptoms may be those of a vague aching pain in the elbow with persistent or recurrent effusion and locking. If locking is not present, at times a clicking sensation will be noticed. As the condition progresses there occurs limitation of motion with increased swelling and pain. At times a palpable loose body may be felt. The diagnosis characteristic for this condition is readily made by roentgen examination. Treatment - The condition calls for surgical removal of the free body or bodies Case History R D.F., age 17, admitted to the hospital on 6-18-35 because of pain and inability to extend the right elbow. Patient stated that two years previously he began to notice that he was unable to extend the right elbow, this gradually increased to the present flexion contracture of 40 degrees No history of injury except the usual trauma of athletes, played four years of high school football The patient recently noticed locking of his elbow at 90 degrees flexion. Examination right elbow, flexion and extension range 140-45 degrees, pronation and supination unrestricted, elbow slightly enlarged and tenderness over radial humeral articulation X-rays a loose body in the anterior compartment of the elbow with a defect on the lateral surface of the capitellum of the humerus Treatment operation on 6-22-35, through a posterolateral incision the joint was entered, 3 loose bodies were found and removed, one measured 1 cm in diameter, the other 2 were very small. Follow-up observation on 9-3-35, there was still present a marked limitation of extension, flexion-extension range 150-45 degrees, pro- and supination normal, no further locking was experienced (Figs. 6 and 7).

Osteochondritis of the Capitellum -- Panner's Disease. This condition was first described by Panner of Copenhagen in 1927.[18] This condition is usually found in young males. There is usually a history of mild preceding trauma or a slight, but repeated trauma to the elbow. The discomfort is usually not severe and accompanies attempts of extreme flexion, extension, and at times rotation Extension is limited in practically all of the cases. X-ray signs consist of an irregularity or fragmentation of the contour of the capitellar epiphysis, except in the early cases the epiphysis is diminished in size due to the changes present in



Fig 6 R D. F Preoperative x-rays showing a loose body in anterior compartment with a defect on the lateral surface of the capitellum of the humerus.



Fig 7. R D. F Postoperative x-rays after removal of the loose body, but still showing the defect in the capitellum of the humerus

the epiphysis The bone is composed of intermingled condensed and rarefied areas. The normal trabeculation is lost. It may simulate osteochondritis dissecans of the capitellum, however, the latter disease does not involve the whole of the capitellum and occurs at a later age. Rest and immobilization is the treatment of choice.

REFERENCES

1. Murphy, H S., and Hansen, C. G. Congenital Humeroradial Synostosis. J. Bone & Joint Surg. 27 712-713 (Oct., 1945).
2. Steindler, Arthur Orthopaedic Operations. Springfield, Ill., Chas. C. Thomas, 1940.
3. Campbell, W C. Operative Orthopaedics St. Louis, Mo , C. V. Mosby Co., 1940.
4. Mouchet, A , et Saint-Pierre, L. Ankylose congenitale hereditaire et symetrique des deux posides Rev. d'Orthop 18 210 (1931).
5. Habbe, J. E., Patella Cubiti. A Report of Four Cases Am J. Roentgenol 48 513-526 (April, 1942).
6. O'Donoghue, D H. and Sell, L. S. Persistent Olecranon Epiphysis in Adults J Bone & Joint Sur 24 677-680 (July, 1942).
7. Henderson, M S., and Jones, H. T. Loose Bodies in Joints and Bursae due to Synovial Osteochondromatosis. J Bone & Joint Surg 5 400-424 (July, 1923).
8. Jones, H. T. Loose Body Formation in Synovial Osteochondromatosis with Special Reference to the Etiology and Pathology J Bone & Joint Surg. 6 407-458 (Apr., 1924)
9. Girdlestone, G. R Tuberculosis of Bone and Joint Oxford Univ. Press, 1940
10. Cleveland, M. Surgical Treatment of Joint Tuberculosis. J. Bone & Joint Surg. 21 607-618 (July, 1939) - also his Surgical Treatment of Joint Tuberculosis. Surg., Gynec. & Obst. 61 503 (1935)
11. Smith, A. DeF. The Early Diagnosis of Joint Tuberculosis. J. A. M. A. 83 1569-1573 (Jan 21, 1928).
12. Milgram, J E. Diagnostic Inaccuracy in Tuberculosis of Bone and Joint and Bursa. J. A M. A. 97 232 (July 25, 1931).
13. Rollier, A. The Conservative Treatment in Surgical Tuberculosis of the Lower Extremities. J Bone & Joint Surg 12 733-748 (Oct., 1930).
14. Bennett, G. E Shoulder and Elbow Lesions of Professional Baseball Pitchers. J. A. M.A. 117 510-514 (Aug 16, 1941).
15. Crysler, W. E., and Morton, H S. Osteochondritis Dissecans of the Supratrochlear Septum of the Humerus. Am. J Roentgenol. 54 41-46 (July, 1945).
16. Hirsh, I. S. The Supratrochlear Foramen, Clinical and Authropological Considerations Am. J. Surg 2 500-505 (1927).
17. Phemister, D B The Causes of and Changes in Loose Bodies arising from the Articular Surface of the Joint J Bone & Joint Surg. 6 278-315 (April, 1924)
18. Panner, H J. An Affection of the Capitulum Humeri Resembling Calvé - Perthes' Disease of the Hip. Acta Radiol. 8 617-618 (1927).

March, H C Osteochondritis of the Capitellum. Panner's disease Am. J of Roentgenol. 51 353-357 (March, 1944)

Miller, L. F Osteochondritis Dissecans of Capitellum of Humerus. Radiology. 27 237-239 (Aug., 1936).

Course No. 9

THE SHOULDER

Lecturers

John Fahey, M.D , Anatomy of the Shoulder

David M. Bosworth, M.D , Differential Diagnosis and Treatment of Common Lesions of the Shoulder

ANATOMY OF THE SHOULDER

John Fahey, M.D

IN THE LAST publication of the *Lectures of the Instructional Courses of the American Academy of Orthopaedic Surgeons*, Milgram[1] gave a thorough review of the anatomical structures about the shoulder Bosworth,[2] McLaughlin,[3] and Ray,[4] in separate articles, have emphasized certain anatomical features in discussing lesions and their operative approach, in the lectures of 1944. Because of the thoroughness in which the subject in general has already been covered, and the renewed interest in the capsular structures since Bankhardt described labrum tears in recurrent dislocations of the shoulder, I shall review the capsular structures and their associated bursae. A method of study of the developing bony structures of the shoulder and the technique of embedding specimens for permanent study in plastic will also be discussed.

Only a minor portion of the hemispherical humeral head articulates with the shallow, pear-shaped, glenoid fossa, which is only one-third to one-half the surface of the head. The glenoid is wider below than above and the cartilage is thinner centrally and below, while the articular cartilage of the humeral head is thicker centrally and below.

The capsular ligament of the shoulder is a thin, dense, fibrous membrane which encircles the joint. Its fibers run in various directions, most of them straight, some oblique, and a few in a circular fashion. When the overlying muscles and tendons are removed, it is a relaxed sacular structure, and does not hold the head in contact with the glenoid. It is attached to the bony margin of the articular surface of the scapula, external to the labrum glenoidale. Anteriorly, above the inferior glenohumeral ligament, it attaches away from the articular margin before being reflected on the labrum glenoidale. The subscapularis bursa, between the superior and middle glenohumeral ligament, and the subscapular pouch, between the middle and inferior ligaments, are the regions where the capsule attaches to the neck at a considerable distance from the labrum, before it is reflected onto the labrum. The glenohumeral ligaments in this region are the only source of connection between the labrum and capsule. Superiorly and anteriorly, the capsule is thickened by fibrous bands, which pass from the scapula to the humerus. The coracohumeral ligament is a thick band which is incorporated in the capsule superiorly, and extends from the lateral border of the root of the coracoid to the greater and lesser tuberosity and to the transverse ligament, which connects them. It has a fan-shaped appearance when viewed from in front, while from the rear it appears to be continuous with the capsule. The glenohumeral ligaments are seen from the interior of the joint where they bulge the synovia. The superior glenohumeral ligament is attached medially to the long tendon of the biceps and passes laterally along the side of the tendon to reach the humerus, near the

upper surface of the lesser tuberosity. The middle glenohumeral ligament reaches from the anterior edge of the glenoid at the lower border of the subscapularis tendon to the lower portion of the lesser tubercle. The inferior is fixed to the scapula anteriorly, below the middle glenohumeral ligament, and passes downward to the under surface of the humeral neck. The middle glenohumeral ligament is not always present, and in such instances the opening of the subscapular bursa is large and is bounded inferiorly by the inferior glenohumeral ligament. Posteriorly, it is firmly attached to the bone and the external surface of the labrum, appearing to be continuous with the labrum, when viewed internally. Inferiorly, it is attached to the origin of the triceps. Superiorly on the humerus it is fixed to the anatomical neck of the humerus and to the transverse ligament of the shoulder, close to the articular surface. Below, it descends upon the humerus for nearly one-half inch from the articular margin, and some of the deeper fibers are reflected upward, toward the articular edge, forming a fibrous investment of the humeral neck. (Figs. 1 and 2.)



Fig 1 Dissection of right glenoid cavity and its associated structures 1) Long tendon biceps, 2) Coracohumeral ligament, 3) Superior glenohumeral ligament, 4) Subscapularis bursa, 5) Middle glenohumeral ligament, 6) Subscapular pouch, 7) Inferior glenohumeral ligament, 8) Subscapularis tendon, 9) Labrum glenoidale

The synovial lining of the articular capsule of the shoulder herniates into various places to produce bursae. These bursae are regarded by some as extensions of the joint, rather than true bursae [5] The subscapular and bicipital bursae are important and well defined. The subscapular bursa is a protrusion of the synovial membrane through an opening in the anterior part of the capsular ligament, near the root of the coracoid process. It extends between the subscapularis muscle anteriorly and the medial portion of the capsule, and the anterior portion of the scapular neck posteriorly. Its connection with the joint is smaller and more tubular than the bursa itself. The bursa may occasionally extend over the tendon of the subscapularis and be continuous with the coracoid bursa. The opening in the capsular ligament, between the superior and middle bands, forms the mouth of the subscapularis bursa.



Fig 2 Left shoulder, anterior view, with subscapularis muscle displaced laterally. The subscapularis tendon 1), and the back of the superior glenohumeral ligament 4). There is a localized, encapsulated, calcified mass in the region of the latter.

Another constant outpocketing of the articular capsule synovial lining is along the biceps in the upper portion of the bicipital groove. It lines the osteofascial tunnel and is reflexed back and covers the biceps tendon The infraspinatus, when present, is small and unimportant A bursa may be found superior to the coracoid and between it and the anomalous insertion of the pectoralis minor.[5]

The subacromial bursa is the largest and most important bursa of the shoulder, and has been thoroughly described by Codman.[5] It separates the acromion and the superior deltoid from the tendons that cover the surface of the capsule of the shoulder joint superiorly. Its floor is the major portion of the greater tuberosity, as well as the tendons of the rotator cuff and the bicipital groove Its roof is formed by the under-surface of the acromion, the inferior surface of the coraco-acromial ligament, and the deltoid origin at the edge of the acromion. Its roof and base are in contact and lined with synovial membrane It is a concavo-convex cleft, approximately one and one-half inches in diameter The subacromial bursa in the anatomical position is subdeltoid, while in abduction it is largely subacromial In hyper-extension and internal rotation it is in the sub-coracoid region. The coracoid bursa may be completely separated from the subacromial, but more frequently, the connection is narrowed by a double fold of membrane A connection between this and the subscapularis is unusual Occasionally a union occurs superiorly on the surface of the subscapularis muscle, the coracoid process, and combined origins of the tendons of the short head of the biceps and coraco-brachialis

The transverse humeral ligament extends between the two tuberosities, curving the bicipital groove, and is covered by thin expansions of the capsule

The labrum glenoidale is a rim of fibro-cartilage, triangular in shape on cross section, surrounding, and deepening the glenoid socket It is wider above and below than at its sides Important studies by Bost and Inman[6] on the capsular structures showed that it is attached loosely to the articular cartilage internally and firmly to the outer edge of the glenoid rim by dense fibrous tissue The outer surface approximates the capsular ligament, while the internal surface articulates with the humeral head The biceps tendon is continuous with the glenoid ligament superiorly, dividing and sending fibers on both sides. The labrum is less firmly attached on the superior portion of the anterior margin

In addition to the usual anatomical studies, the bursae about the shoulder have been studied in various ways Injections with radio-paque substances and roentgenographic observations are suitable for clinical studies Injected melted wax had been found to be brittle and its finer extensions are likely to break

Vinylite is easy to use, but its shrinkage is considerable, and it is likewise very brittle I have found Wood's Metal (fusible alloy with a melting point of 72-74°C) the most suitable material for outlining bursae, and being durable, so that its pattern is maintained. It should be remembered that any injected mass distends and distorts somewhat the true anatomical picture, but their extent and communications can be thoroughly demonstrated in this fashion A shoulder is removed at autopsy, and a small bulbus-end glass rod, with purse string suture, is tied in place in the anterior capsular region and connected to a funnel by rubber tubing (Fig 3) The specimen and its attachments, including the lower three-quarters of the funnel, is placed under water at 170°F for 10 minutes, at which time melted Wood's Metal is poured into the funnel The capsule with its bursal communications are clearly outlined, and a permanent metal mold can be secured The subdeltoid bursa is outlined by a similar study. (Figs. 4, 5, and 6)

Recently a considerable amount of work has been done on embedding anatomical specimens in plastics. Castolite has been found to be the most simple to use for thin specimens It comes as a thick, viscous liquid, with a hardener A base or layer is first poured, on which the specimen is to rest, and this gels at room temperature, or more rapidly by setting the container in a pan of water at 125°F The specimen is positioned just before gelling has occurred, and then it is allowed to continue until the gel becomes fairly rubbery The specimen is then covered with Castolite and permitted to harden at room temperature. The surface is then covered with cellophane before the last heating It is advisable when using wet specimens, to dry them as much as possible, with paper towels or filter paper, before starting the procedure Thin, gross anatomical specimens and brittle corrosive preparations

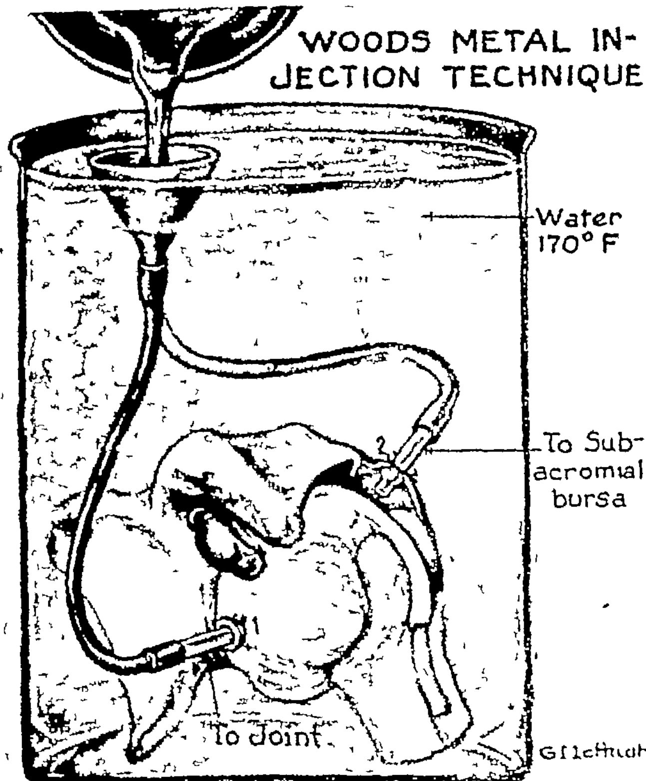


Fig. 3 Cannulae tied in shoulder capsule 1) and subacromial bursa 2) and connected with a funnel The specimen and lower portion of the funnel have been placed under water at 170°F for ten minutes prior to pouring the Wood's Metal.



Fig 4 Wood's Metal injection of shoulder into capsule, (as in Fig 3) 1) its connection, 2) the subscapularis bursa, and 3) its outpocketing

are particularly suitable for this method

A study of the ossification centers about the shoulder, and particularly the spine, is best accomplished by means by staining and clearing methods. Haviland[7] has modified the well-known Schultze and Mall method and obtained very satisfactory results. Its simplest and most useful application is in the embryonic and fetal stages of development, as the skin at this time is more transparent Evisceration of the specimen will prevent masking of the skeleton by the abdominal organs. Acid fixatives are not satisfactory, as they remove the calcium which the Alizarin stains. This applies particularly to specimens that have been in formaldehyde for long periods of time, where the formaldehyde has turned acid in reaction. A short period of formaldehyde fixation, or 70-95% alcohol, is preferable. The shrinkage that occurs in alcohol will disappear in the potassium hydroxide, or clearing solution. After fixation, the specimen is soaked overnight in water to remove the fixative, and is transferred directly to 3% KOH solution. 3% is the usual optimum concentration, but in fresh material or delicate embryos, 1% or even $\frac{1}{2}$% is desirable. After 24-48 hours in KOH, it is transferred to full-strength hydrogen peroxide for bleaching. The specimen is again transferred to 3% KOH and set in the sunlight if possible, as this seems to accelerate the process. The KOH solution is changed when it is discolored. When the bones can be clearly seen through the tissues, the stain is added directly to the KOH, or clearing solution. The stain is a saturated solution of Alizarin in 95% alcohol, which is added to the KOH or clearing solution, to the desired intensity. After thorough staining for from 8-12 hours, the specimen is transferred to Mall's solution, which consists of equal parts of 3% KOH and 10-20% glycerine. This solution is changed on discoloration until only the bones remain discolored. The specimen is transferred to 50,



Fig 5. Wood's Metal injection of subacromial bursa 1) and of capsule 2)



Fig 6 Wood's Metal injection of subacromial bursa 1) and of capsule 4) with its connections, bicipital groove extension 5) subscapularis bursa 7) coracoid extensions from subscapularis bursa 2) and 3)

75, and 100% glycerine, remaining several days in each, and is stored in pure glycerine with thymol crystals to prevent mold growth Specimens that appear not too clear at times, become transparent and satisfactory after they are transferred to glycerine

REFERENCES

1 Milgram, J E Shoulder Anatomy Lectures on Regional Orthopedic Surgery and Fundamental Orthopedic Problems, Ann Arbor, Michigan, J W Edwards, pp 55-68

2 McLaughlin, Harrison L Muscular and Tendinous Defects at the Shoulder and their Repair, Reconstruction Surgery of the Extremities, J W Edwards, Ann Arbor, Michigan, 1944, pp 343-358

3 Bosworth, David M Muscular and Tendinous Defects at the Shoulder and their Repair, ibid, pp 380-390

4 Ray, Bronson S Injuries to the Nerves about the Shoulder, ibid, pp 371-376

5 Codman, E A The Shoulder Boston, Mass Thomas Todd Company, 1934

6 Bost, Fredrick C, and Inman, Verne T. The Pathological Changes in Recurrent Dislocation of the Shoulder, pp 595-613, J Bone & Joint Surg, V 24 595-613 (1942)

7 Personal communication from Dr Thomas Haviland

DIFFERENTIAL DIAGNOSIS AND TREATMENT OF COMMON LESIONS OF THE SHOULDER

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Difficulty in the diagnosis and treatment of lesions of the shoulder arises from two main factors first, the mental hazard of approaching a region which has always been thought of as difficult of repair and where great uncertainty lies as to production of favorable results, secondly, the actual difficulties arising from the presence of deeply buried tissues causing pathological response, paucity of demonstrable objective clinical signs in many instances, and instability of actual articulating surfaces. We have spoken on previous occasions of an important group causing shoulder disability, namely pathology related to the short rotator cuff While we shall entirely disregard this form of pathology in the present paper, we strongly urge that the reader refresh himself as to the varied picture and multiplicity of these defects by referring to such previous reports. We have also written at considerable length, albeit with much haste, of nontraumatic lesions causing shoulder disability. Since that report amplifies the present discussion, I would refer the reader to it in the printed report of the *Lectures of the Instructional Course* of 1946.

At first thought one might conceive that the present discussion duplicates the last-mentioned paper. There is, however, a clear-cut difference between enumeration of nontraumatic lesions of the shoulder, and an attempt to guide the reader toward accuracy of diagnosis in such pathology. In the present report we shall take this latter course, at times perhaps becoming somewhat wearisome with elementary discussions Might it not be wise, however, to point out that success stands on review of basic facts and minutiae? They cannot be reviewed too often

All lesions about the shoulder can be classified in one of two ways. From a structural standpoint they must be related to the bodily system the pathology occupies. They may be osseous, cartilaginous, muscular, tendinous, ligamentous, capsular, synovial, neurological, vascular, or dermal. In etiological classification structural elements are disregarded. Instead the lesions are grouped as to their original cause. They would, by this method, be grouped as congenital, trophic, metabolic, developmental, neoplastic, and infectious.

Having accepted such classifications, one would believe, at first glance, that they provide an outline from a diagnostic standpoint. Actually such classifications are of no help whatever in diagnosis but are merely convenient pegs on which to hang the finished product For the present discussion let us forget them and start afresh.

Table I

Diagnostic Aids	
Knowledge of the Normal	Roentgen Findings
History	Laboratory Data
Past History	Confinement Observation
Physical Facts	Exploration
Physical Examination	

The well-versed clinician recognizes immediately many shoulder lesions. The point is not that he recognizes them, but that the observer fails to realize that he recognizes them through an intricate process of observation and correlation of facts, which his training enables him to perceive almost at a glance. That he has previously seen cases that appeared similar does not lead him to accuracy of perception of the etiological factors involved. The "snap diagnosis" is often not the correct diagnosis, whereas the rapid diagnosis based upon the almost instantaneous grasping of many facts may be accurate. It may sound pedantic to state that one must mentally trudge through the knowledge of the normal, a history, past history, physical facts, physical findings, roentgen findings, laboratory data, confinement observations, and, finally perhaps, exploration, to arrive at a proper conclusion. Observe the successful clinician and one will perceive that he does just that. The flow of shoulder cases under examination may continue smoothly and rapidly, until some slight variation in pattern of facts occurs. Then the whole process of such a physicians's procedures must stop, and the lengthy road of

return to elementals must be traversed before a final correct conclusion may be achieved

Knowledge of the Normal

I daresay that many of us have not thought since medical school days of how important a knowledge of the normal may be, yet the words "know thyself" were uttered a long time ago! At the starting point, the very essence of clinical perception, the one single thing that takes the greatest training and shows the most developed clinical experience is to perceive and know normalcy in all its variations. In the following illustrations let us forget, or minimize, if you will, the actual definite clinical picture that may be presented. Such illustrations as are shown will be to emphasize one basic factor in the chain of a diagnostic problem, rather than a complex entity itself (Fig 1A) The normal has many patterns, of which the illustration shown is merely one. No essential abnormalities exist therein, though an apparent variation of the whole might suggest pathology It should surprise no one that the development of a complex shoulder apparatus, part of a human being arising from the union of two cells, should not always appear the same. Let this roentgenogram, if you will, lead your mind, admittedly broadly trained in matters of this nature, to translate your perception of the film into a flesh and blood patient. Translate the roentgen findings into clinically perceptible changes which can be explored by examining senses other than the eye, such as the tactile, auditory, etc. Imagine this patient before you! Accept the fact that this individual may be thin or obese, large or small, etc., but that this shoulder functioned normally, that it was asymptomatic and disclosed, aside from the visual shadowgram, no pathology whatever It then represents the normal. The normal may exhibit many forms and innumerable peculiarities. As our recognition of the normal develops, we can for the first time discern the presence of pathology.

History (Onset and Duration, Treatment to Date, Reaction to Treatment)

The elementary clinical approach invariably includes the taking of a history. Medical reaction to this procedure generally passes through three stages. The young man evinces moderate interest, at a later date boredom ensues, and finally great interest and exactitude is acquired. Interest, precision, and speed are acquired as experience dictates a routine to be followed. This is so for all the diagnostic factors given hereafter. As to history, the routine may be simple. Noted should be time and method of onset of the lesion and its duration Modes of treatment applied to date, and the patient's reaction to treatment, will practically complete the picture. Onset and duration (Fig. 1B) may be illustrated in the patient with productive changes about the upper border of the greater tuberosity of the humerus Many lesions produce such changes. Given the history of slow steady progress of discomfort over a period of many years, one's attention is directed to the possibility of traumatic change produced by heavy abnormal occupation.

Treatment to Date (Fig. 1C)

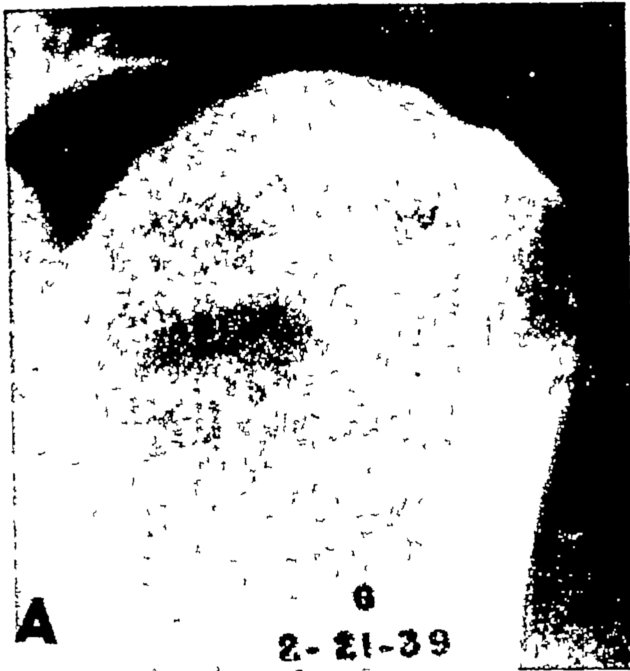
When one considers that all forms of physiotherapy have been employed in this patient and that finally accurate and efficient needling of the defect in the supraspinatus tendon shadow had been done without alteration thereof, one cannot but consider the possibility that the dense lesion shown was osseous instead of calcific, an accurate observation. Proper treatment of excision would then be recommended.

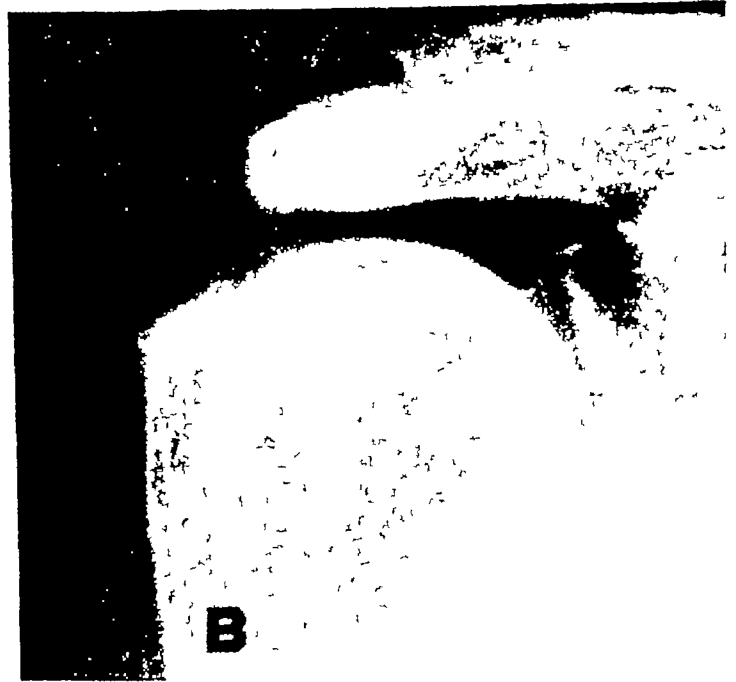
Reaction to Treatment (Fig. 1D)

Though intensive roentgen therapy has been administered to this patient's scapula, the lesion has progressed. This fact, of course, is indicative of a malignant tumor of the type that is resistant to roentgen therapy, namely a sarcoma.

Past History (Injuries, Operations, Illnesses, Heredity, Contacts, or Occupations)

Important in itself, and not even secondary to history, are the influences of past events on the individual. Knowledge of injuries, operations, and illnesses must be carefully investigated (Fig 2A) This patient shown with an amputation of the upper arm complained of continuous pain in the shoulder region overshadowing phantom arm symptomatology. She disclosed a past history of hysterectomy, artificial menopause, and a great deal of symptomatology associated therewith, but her main complaint was shoulder pain. On the basis of





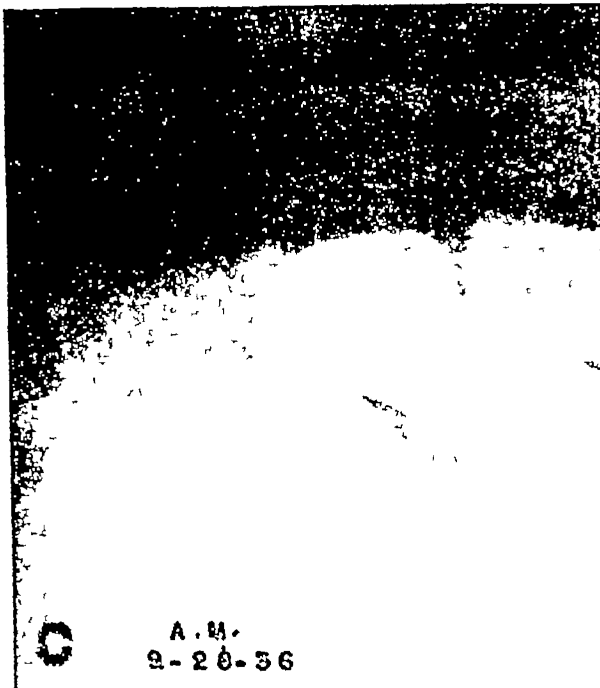




Fig 1

her past history, referral for estrogenic substance resolved her status to one of asymptomatic and bearable phantom arm symptomatology

Hereditary factors disclosed in the past history may be important from a diagnostic standpoint even where advice as to treatment is apparent (Fig. 2B). Congenital elevation of the scapula is a satisfactory instance. That it can frequently be controlled by extensive surgery is herewith apparent.

Contacts or occupations in the past, if

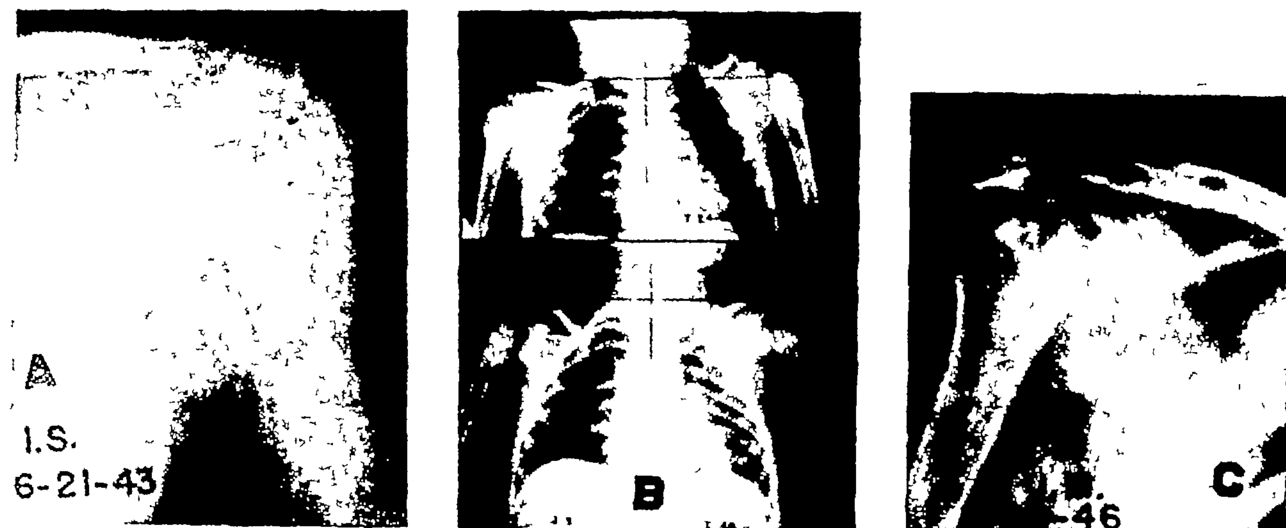


Fig 2

known, often are the final illuminating points in diagnosis (Fig. 2C). One's attention is frequently drawn to the possibility of a tuberculous etiology by the past history Efficient arrest of the lesion by any one of numerous methods of arthrodesis may be promoted.

Physical Facts (Age, General Nutrition, Reaction to Shoulder Lesion)

This includes a group of factors of broad range immediately appreciated but generally not considered as being specifically noted. The age of the patient often leads one to include or exclude certain pathology without realizing that one has done so (Fig 3A). Note here a young boy with a mass on the scapula. Malignant neoplasm is known to be present by other data obtained. It is practically certain that it will not be carcinomatous, for instance, because of the age factor. It was sarcomatous.

General nutrition at time of examination similarly excludes certain pathology (Fig. 3B) Here we find a patient with fairly good general nutrition despite known malignant lesion of long duration. A lesion such as hypernephroma would be immediately ruled out. She had a chondrosarcoma for which interscapulothoracic amputation was performed.

Reaction to the shoulder lesion present often helps in the diagnosis as to involvement of other structures than those immediately appreciable (Fig. 3C). This child, while depressed and discouraged by lack of function of the arm, showed no great evidence of painful phenomena such as would be produced by periarticular adhesive changes, etc

Physical Examination

Table II

Physical Examination		
Motion - Protection		Heat
Power - Control		Swelling
	(atrophy	Crepitus
Musculature	(spasm	Tenderness
	(atony	Deformity
Sensation	(superficial	Audible phenomena
	(deep	

We shall rapidly move over this portion of the diagnostic procedure It has been stressed time and again in the past. We shall briefly review the findings that should be made. They are important. Ranges of motion, evidences of pain on motion, and protection or spasm during mobility or at the extremes of mobility must be appreciated. The presence of absence of power and its accurate application (control) must be carefully checked throughout the complex musculature of the region Muscle spasm, atony, and atrophy notations will take considerable time if completely and accurately done. The intactness of sensation, both superficial and deep, is important. The presence of heat,

swelling, tenderness, and crepitus are all rapidly disclosed to the sense of touch. Deformities are visually perceptible. Even audible phenomena may be present, as in those occasional subscapular lesions where the shoulder may be heard to crunch and crack from across the room without apparent painful stimuli.

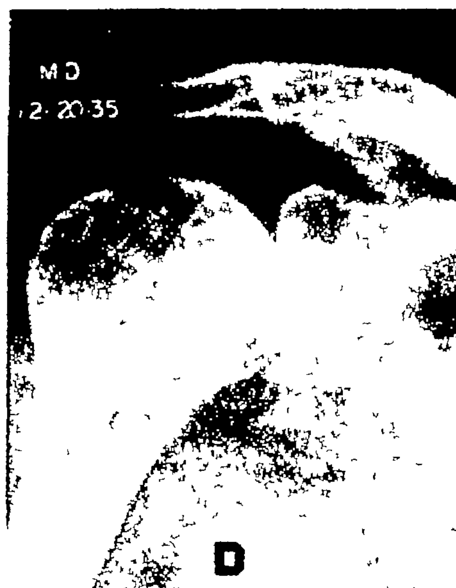
Roentgen Findings

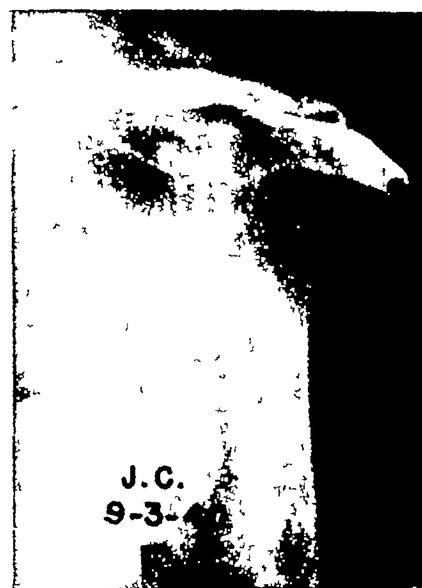
We will divide these findings into those in the osseous structures and those in the soft tissues. Variations from the normal denoting pathology in the osseous structures basically consist in the following changes. VARIATIONS IN OUTLINE should be noted (Fig. 4A). They are not always as gross as in this instance. One should stop here and again recall that knowledge of the normal is important and takes time and experience to acquire. Here we have a distorted outline with unbroken cortical shadow; therefore, a tumor, though probably benign. The reader's diagnosis of osteochondroma is correct. Surgical removal, while difficult in such an extensive case, is possible. INTERNAL OSSEOUS STRUCTURE change is not always as readily appreciable as in this instance (Fig. 4B). It may be coarsened, altered in structure, destroyed, or changed in numerous ways. One's diagnosis of malignant lesion in this instance is not based upon the internal structure alteration alone. Visualization of ACCESSORY BONE PARTS frequently establishes the basis of mechanical difficulty (Fig. 4C). Here we note an apparently congenitally unattached acromial margin. Surgical fixation to the acromion resulted in disappearance of symptomatology. DENSITY may be altered either as to increase or decrease (Fig. 4C). Here we have the moderate generalized atrophy associated with generalized periarticular adhesive phenomena of the shoulder joint. The SIZE of the bony parts may vary, either with increase or diminution, local or general (Fig. 4E). This patient with gigantism (pituitary type) had difficulty not only with his shoulders but with all his joints. RELATIONSHIP of the bony structures to each other should be noted (Fig. 4F). Frequently changes in relationship are minimal and yet diagnostic. Pathological dislocation of this shoulder is gross. Notation of change at much earlier date might have favored reconstruction. The presence or absence of EPIPHYSEAL



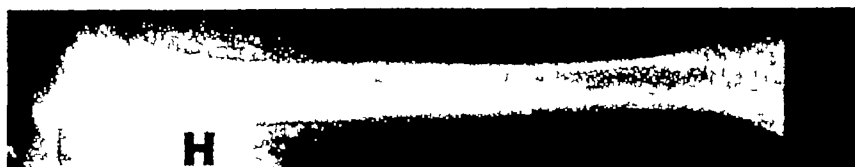
Fig 3











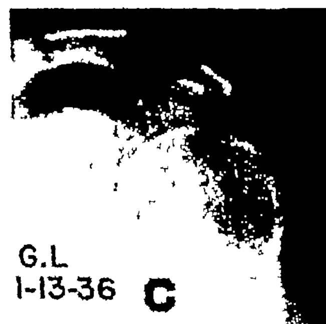






Fig 4

Table III

A Roentgen Findings

Osseous	Soft Tissues
Outline	Swelling
Internal Structure	
Accessory Bony Parts	Atrophy
Density	
Size	Obliteration of Fat Densities
Relationship	
Epiphyseal Lines	
Reaction Area	

LINES and their appearance, are important (Fig. 4G). By denoting the age of the patient they tend to focus attention on proper etiological consideration. This pathological fracture of a cyst of the humerus, properly diagnosed, was treated surgically with curettage and bone graft to a successful conclusion. Perhaps the most difficult roentgen finding in osseous structures is the delineation of a REACTION AREA. Such an area is the earliest finding in osteomyelitis. It depends upon the replacement of normal material in the bony interstices with other material of higher specific gravity. This tends to bring the density of the interstices up to that of the reticulum and smooth out or "haze" the bony area involved (Fig. 4H). We fear this illustration may be too small to appreciate the reaction area. It is clearly seen in the original roentgenogram in the proximal two thirds of the humeral shaft.

Soft tissue changes such as soft tissue swelling are frequently present in the roentgen film but often not reported (Fig. 5A). This tubercular shoulder lesion shows soft tissue swelling in the axillary region from abscess mass Not only is it of diagnostic significance, but of prognostic significance as well. Final arrest of the lesion with shoulder arthrodesis was obtained. SOFT TISSUE ATROPHY should likewise be appreciated in the roentgen film as in this instance of myositis ossificans (Fig. 5C) Obliteration of NORMAL FAT DENSITY MARKINGS occurring between soft tissue structures is frequently not noted (Fig 5B). It can be of extreme significance and should always be looked for. This patient with a frozen shoulder subsequent to an inflammatory condition illustrates this finding.

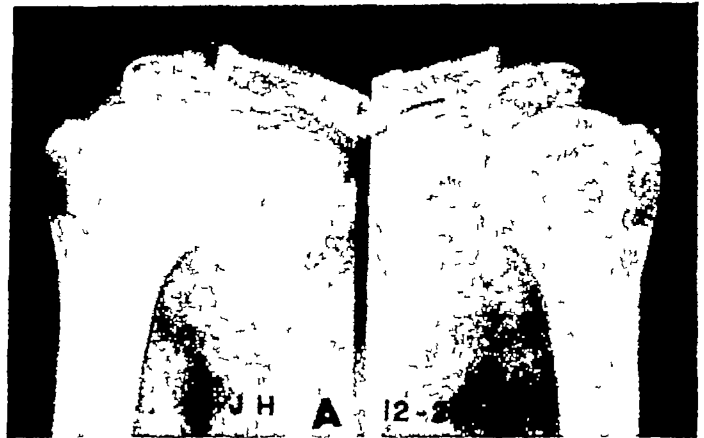






Fig. 5

Laboratory Data

Urine analysis is difficult to correlate with shoulder disability (Fig. 6A). As one extends

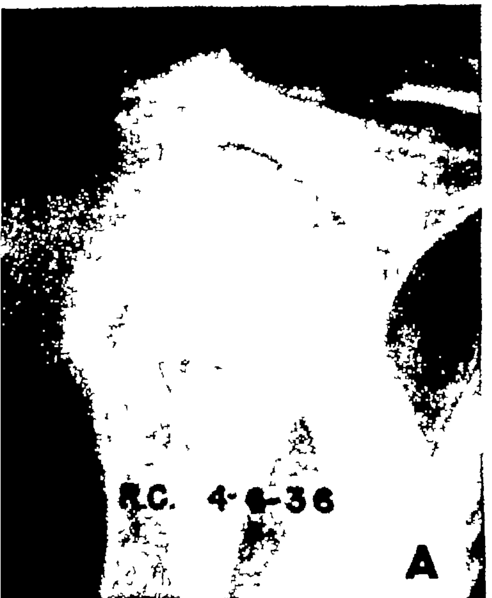












Fig 6

Table III

B Laboratory Data	
Urine Blood Wassermann Sputum Sedimentation Rate	Calcium - Phosphorus - Phosphatase Uric Acid Agglutination Tests Electrocardiogram

urine analysis to centrifuged specimen examination, for the tubercle bacillus, however, a procedure which clinched suspicion into certainty in this instance, its importance becomes apparent. Blood count as an indication of general bodily reaction is more readily accepted in discussion of classification of shoulder lesions (Fig 6B) In this instance of simple giant-cell tumor of the upper end of the humerus, one indication of the absence of malignancy were the normal red blood cell and hemoglobin findings In a disintegrating nonpainful shoulder, Wassermann examination becomes important (Fig 6C) Here, in an instance of generalized aseptic necrosis, incident to osteochondritis (occurring several months after trauma), a negative Wassermann finding became important Sputum examination in a suspected infected shoulder lesion established the diagnosis in this patient. (Fig. 6D) Sedimentation rate in an acromioclavicular lesion aided not only classification as to etiology but directed attention to the activity of the lesion as well (Fig. 6E). Calcium, phosphorus, and phosphatase studies disclosed a parathyroid tumor basis for the changes noted not only in the shoulders but throughout the skeleton of another patient. (Fig 6F). Uric acid determinations, and agglutination test for melitensis and other similar lesions, may prove of importance. Electrocardiogram studies and other physical laboratory data are frequently of great help. A patient in a tuberculous institution complaining of shoulder symptomatology had severe cardiac pathology. Adhesive periarticular changes incident to the cardiac pathology were present and not a low-grade tuberculosis sicca.

Confinement Observation (Residents and Nurses, Reaction to Treatment, Consultation with Other Specialists)

In shoulder lesions, as elsewhere, complete accurate determination of pathology present may depend upon hospital observation In the main, data can be secured through this method in three ways. The reports of nurses and residents as to the activities and suffering of patients provide excellent data (Fig 7A) In this patient with a fracture of the humerus, extreme pain beyond that usually present was reported by the resident, demanded immediate attention, and actively drew attention to the pathological nature of the fracture The lesion was an enchondroma, benign. Decompression by evacuation of the extruded material provided almost immediate relief of pain. Union eventually occurred

The reaction of the patient is best studied

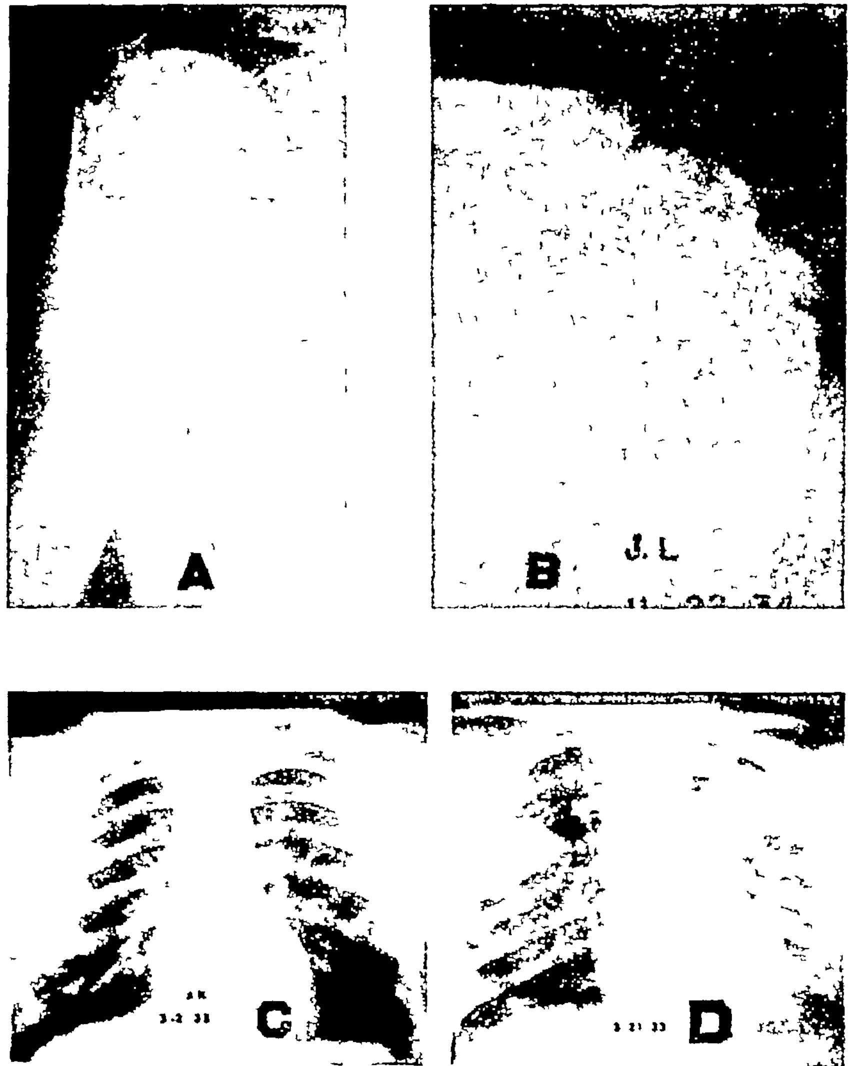


Fig 7

under hospital observation Progress of symptomatology may denote infection or other specific process. (Fig. 7B). In the instance shown, rupture of a very painful calcified supraspinatus lesion gave relief and diagnostic data even before roentgenograms were seen Consultation with other specialists can be most satisfactorily obtained during hospitalization. (Fig. 7C) In this patient chest complications and medical reports first drew attention to malignant lesion involving the shoulder girdle.

Exploration, Commonly Called "Operation"

Exploration is not so frequently a confession of lack of knowledge as a confession of the lack of *exact* knowledge Often it is the only answer to many shoulder lesions Frequently the true pathology present is not appreciated before exploration, and usually the extent of the pathology is not understood. The shoulder can be as rapidly and easily explored as the knee joint, and such exploration need produce no following disability Where doubt exists and where serious disability may ensue, one should not hesitate to open the shoulder any more than one would to explore the knee. Let one be forewarned, however, that even with the shoulder joint exposed, many forms of pathology will not become apparent on exploration without that essential "knowledge of the normal."

Differential Diagnosis

On occasion certain lesions may be extremely difficult of accurate diagnosis until either time has passed or the pathological microscopic section is before one. Differentiation of tumors of the shoulder as benign or malignant has often been confusing Accurate classification of low-grade suppurative infection from tuberculosis, nonspecific granuloma, nonsuppurative inflammatory conditions, and rheumatoid arthritis has not been satisfactorily made at times.

Here is a brief story of a patient with a tremendously large, rapidly growing lesion involving the humeral head and upper shaft. Clinical examination of the patient and the roentgenograms furnished a diagnosis of malignancy. Interscapular thoracic amputation was performed for relief of pain until a supposedly fatal outcome. Simple giant-cell tumor was revealed by histological section and the patient survived (Fig 8). Could one, in such a case, have reasonably foretold the benign nature of the tumor? Perhaps we could have, if we had noted a complete range of diagnostic aids and not been misled by the roent-





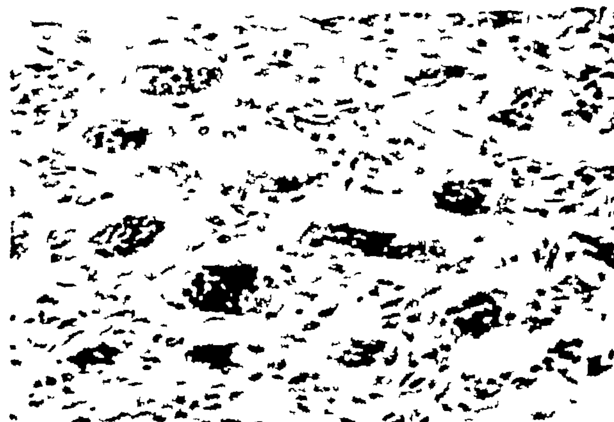


Fig 8

genograms The patient gave a history of considerable duration of the lesion though minimal reaction to treatment. Her hereditary past history was negative and the physical fact of general nutrition was excellent. The general nutrition had not changed during development of the tumor She reacted but little to her lesion until secondary infection became present without actual disruption of the skin Her physical findings and roentgenogram changes were misleading Her blood findings were normal as to red cells and hemoglobin. Consultation with other specialists revealed no malignancy elsewhere, nor any chest involvement. Biopsy in her instance consisted of the interscapular thoracic amputation, justifiable in view of her lack of response to roentgen therapy

This represented an example of expanding local lesion without general bodily reaction. We would have been justified in suspecting a benign lesion preoperatively.

Let us then return to simple fundamentals in diagnosis and to a greater evaluation of the assembled findings as a whole.

Course No. 11

THE THIGH

Lecturer

Joseph E. Milgram, M.D., "The Thigh - Certain Aspects of Applied Anatomy"

CERTAIN ASPECTS OF APPLIED ANATOMY

J. E. Milgram, M.D

AS A CONSEQUENCE of the growth of knowledge of locomotor lesions, we are developing in orthopedics specialists within the specialty. Consequently, not unlike the blind men in the parable each of whom described the elephant differently, each group of orthopedists tends to see the thigh from the viewpoint of its own interests. The orthopedist treating primarily trauma, views the thigh as a dermo-muscular container for a brittle femur and the channel for a vulnerable neural and vascular pathway to the lower extremity. The pediatric-orthopedist sees the thigh as the bearer of susceptible epiphyses and diaphysis with troublesome knee and hip joints traversed by a frequently paralyzed quadriceps. The adult geriatric-orthopedist sees a weak femoral neck, arthritic hip and knee, an easily affected hip abductor mechanism, tendons as the site of degenerative lesions, and a buttock and groin as a track for abscesses

Most of us tend to picture the thigh as a rather solid, sturdy mechanism. In this hour we will try to picture it as containing a weight-sustaining cane or core surrounded by a layered muscular mechanism. Between layers of muscle and their fascia, compartments or spaces exist, knowledge of which facilitates surgical exposures and helps understanding of the geography of massive purulent collections encountered at times in its substance (Fig. 1).

We may hope as well to consider some commonly-met muscular and bony features of the thigh and inferentially relate them to clinical problems for emphasis.



Fig 1 Extensive purulent collection and secondary slough of antero-lateral compartment of thigh following infected gunshot wound of femoral shaft

Where is the thigh when an adult stands with feet together? Note how the hip joints are farther apart than the knee joints Only when standing "At Ease" (Fig. 2) are the centers of hip and knee joints in line and even then the upper two-thirds or three-fourths of the femur is lateral to the line of weight bearing. In a broad-hipped female the thigh slants inward even more View an x-ray plate of the pelvis and hip taken while standing on one leg and consider the stresses the pelvic-trochanteric abductor muscles must resist to hold the super-incumbent body erect over the asymmetrically placed hip. View this hip ankylosed in abduc-

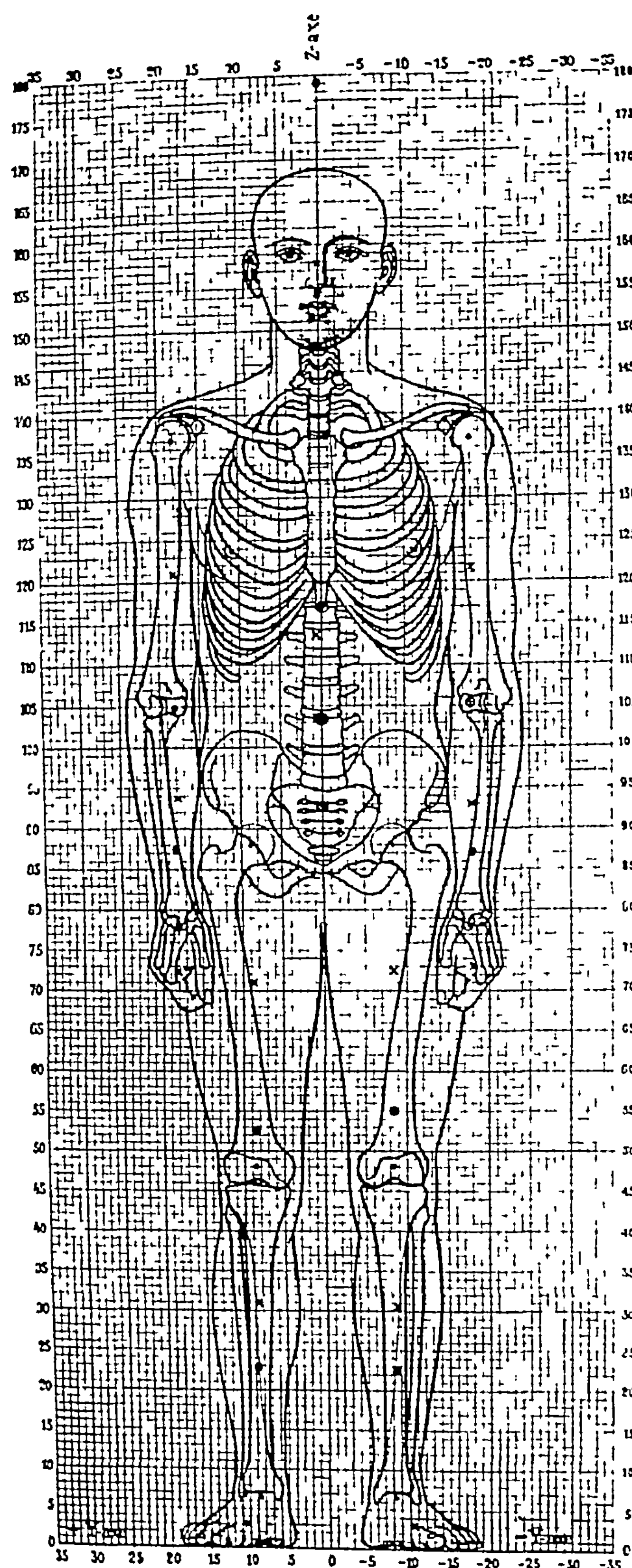


Fig 2 Alignment of (marked) centers of gravity of extremities in standing "At Ease" (From R Fick)

tion and contemplate how seriously it disturbs the maintenance of erect posture in walking. Fixed abduction (bony, or muscular, or fascial) contracture prevents the line of the center of gravity from passing through the thigh in normal fashion to the supporting foot. Stand so, with both heels on a rule. When the heels are a mere seven inches apart lift one foot from the ground. Note how one must tilt his body to the side of the supporting foot. In fact an adduction hip contracture, unless extreme, is far less disabling than an abduction contracture resulting from surgery. It is, accordingly, essential to measure preoperative adduction range in planning an angulation abduction osteotomy to ensure that appreciable hip abduction contracture will not remain

The rigid femoral core has a laterally placed shaft in the thigh. The trabecular anatomy of its curved neck deserves attention even though we no longer believe that it fulfills the simple mechanical requirements for a crane which earlier anatomists heatedly debated. Centrally, the neck bears a capped epiphysis and a head which is larger than its acetabulum and part of which is positioned at approximately right angles to the powerful medial, more strutlike trabeculae (Fig. 3). The epiphysis in childhood holds to the neck bone because fingerlike columns of bone and cartilage cells are lined up side by side every-

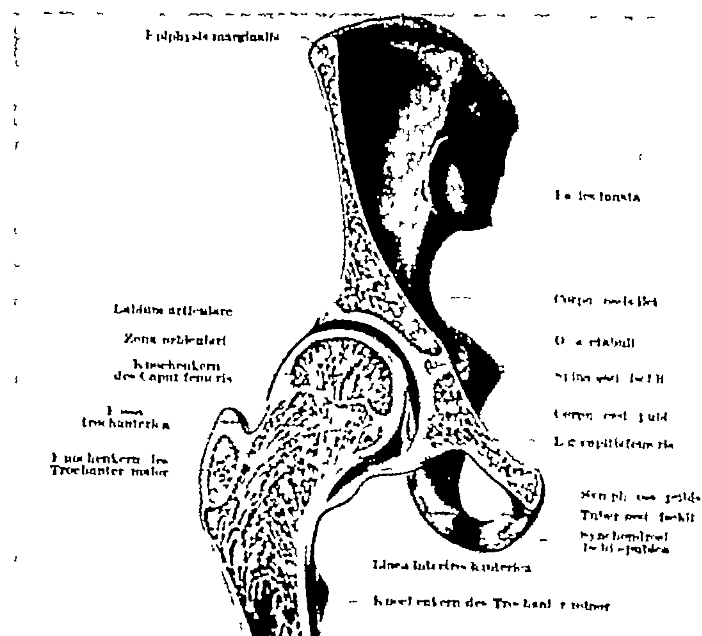


Fig 3 Hip, sagittal section (from V. Lanz-Wachsmith).

where across the epiphyseal line area adjacent to the cartilaginous plate. Let these interlocking little fingers but loose their grip, as when ossification for some hormonal-chemical reason is disturbed, and weight and extension stresses can then produce a slipped epiphysis in a four-year-old hypothyroid cretin as well as in a twelve-year-old Froehlich Since the neck is normally anteverted (abnormal degrees of anterversion in congenital hip lesions have been stressed for seventy years, since Mikulicz' time), the capped epiphysis slips gradually, and at times suddenly as well, backward on the neck so that the anterior edge of the exposed neck may contact the front edge of the acetabulum Manipulative trauma has fused these two contacting structures solidly at times. We are all aware of the vascular problems of the hip (Fig. 4).

nails, and bone grafts are guided readily once the trochanteric cortex is perforated. The shaft is for the most part dense and triangular, and we note on serial cross sections the shape and size of its medullary cavity in relation to possible intramedullary nail fixation. Distally we observe the flared cancellous end of the shaft. Steinmann selected this cancellous bone for his thigh traction nail In adults, he chose a site extending from the adductor tubercle medially to the epicondyle laterally, going posteriorly to avoid traversing knee joint capsule In childhood he went a finger's breadth higher to prevent injury to the epiphyseal plate Incidentally, even in 1911 he had stopped trying to hammer pins through a too-often brittle femur and was drilling pins in place with hand and electric drills.

Draped about the eccentric coursing femur

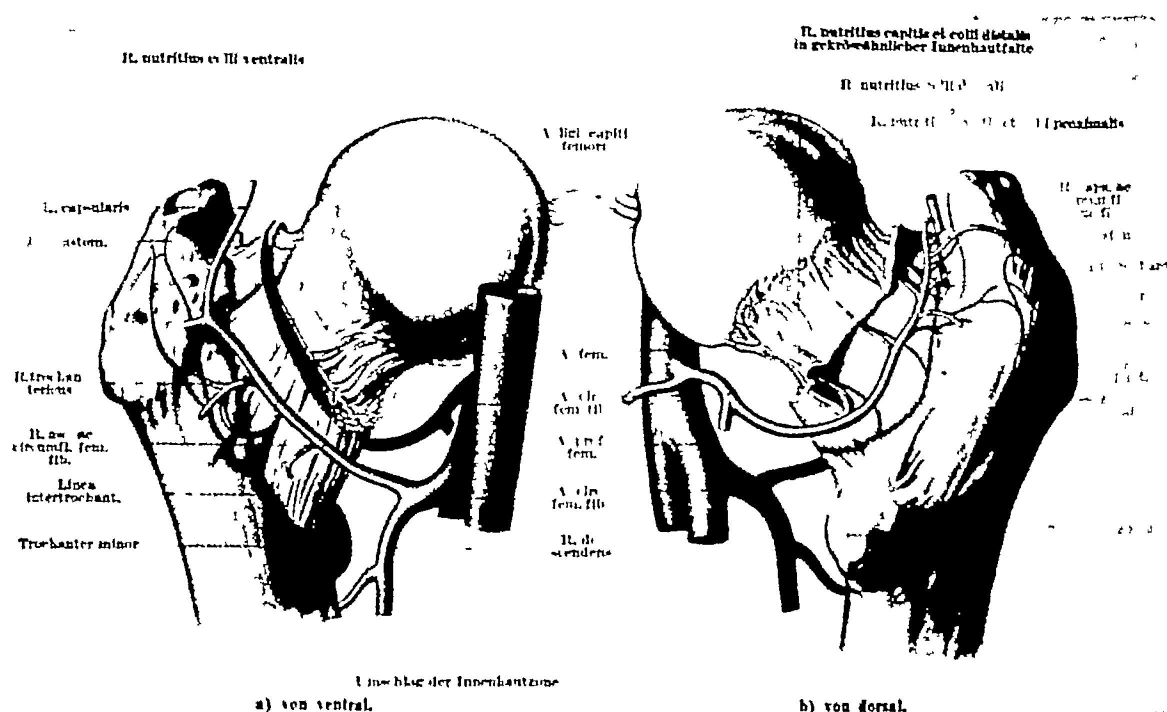


Fig 4 Gross blood supply of neck (from V. Lanz-Wachsmith)

are layers of powerful muscles, many of which must alter length markedly during motion of leg and trunk (sartorius shortens 133 mm., rectus femoris 60 mm.). Accordingly, they are provided with gliding mechanisms of fascia, areolar tissue, and bursae. Injury to these seems to be reparable, else restoration

The lower thigh epiphysis is anchored more securely by four diaphyseal prominences reminiscent of the efficient fixation seen in flexed-knee quadrupeds

The neck of the thigh-bone, we note in passing, has a denser tubular cortex and a softer center through which pins, blade plates, flanged

of athletic abilities would seldom occur.

Embryologically, the deep fascia of the abdomen pouting over the out-pushing lower limb buds thickens into the dense enveloping fascia lata. It is this fascia (Fig. 5) which splits in some place to include muscle bellies It splits to enclose the tensor fascia, which, when improperly coordinated with the gluteus maximus (which also inserts in the fascia) permits the longest fibred fascial tract of Maissiate between them to snap over the greater trochanter in certain phases of hip motion. It includes most of the gluteus maximus and serves as its tendon of insertion to fix the body on the thigh while the hamstrings and quadriceps are lifting the body from the seated to the erect position. The fascia forms a live broad ligament to help stabilize the knee joint in motion. It can pathologically shorten, causing deformity of pelvis on femur and femur on tibia. It also helps form the spaces in the thigh into which we run 1500 cc. of clysis fluid without difficulty. "Subfascialclysis" would be more accurate than "hypodermoclysis."

Contrary to impressions gained from serial cross sections, the thigh is not a compact structure of muscle and bone encased in fascia, subcutaneous tissue, and skin. The thigh contains large and strategic fascial spaces extending almost its full length, traversed only by fine areolar bridges.

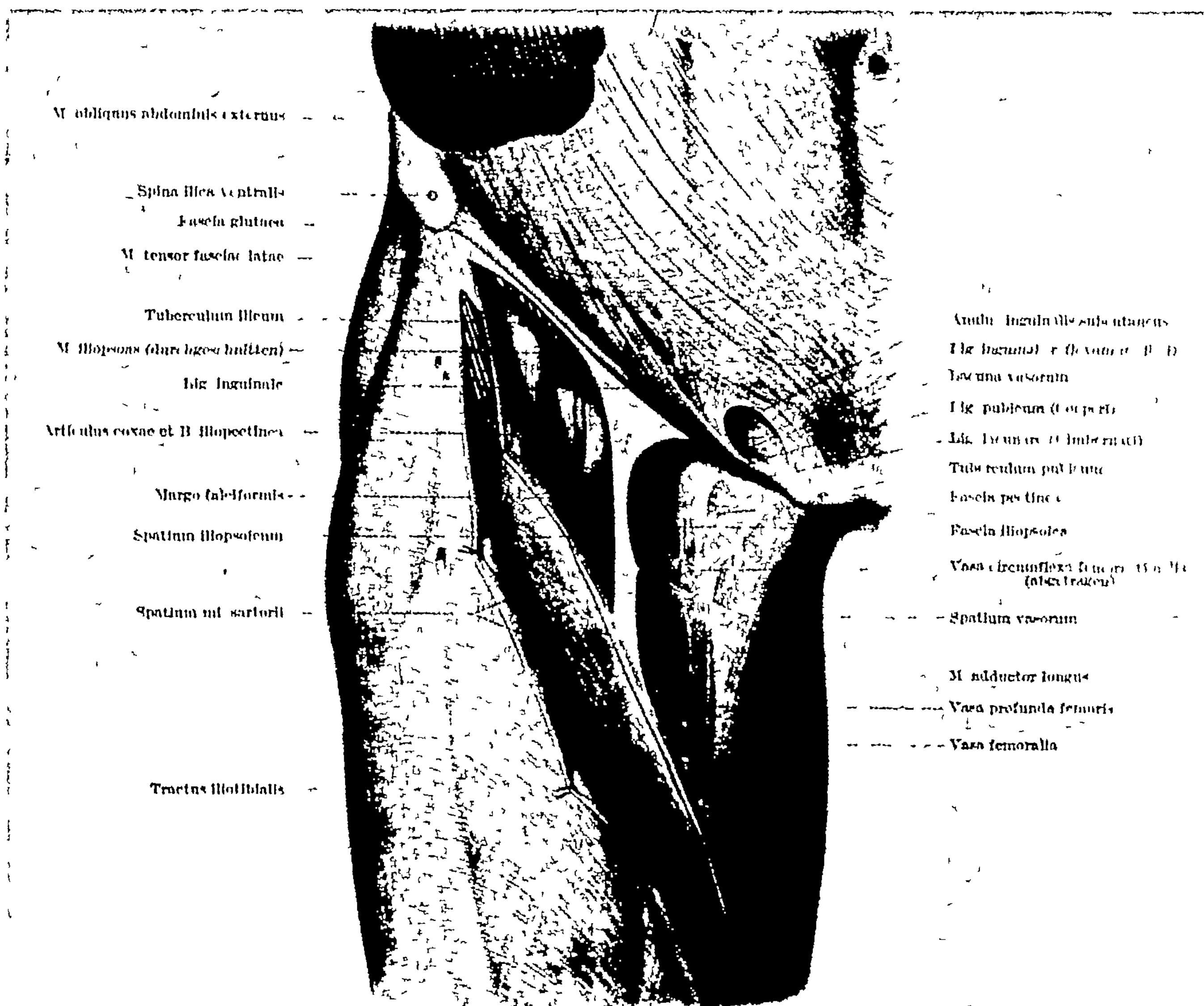


Fig 5 Fascia lata splitting around sartorius Anterior thigh (from V Lanz-Wachsmith)

Thigh Space Anatomy

Tendons are provided with mesothelial sheaths or thecae for freedom of movement The more superficial muscles of the thigh are provided with fascial compartments which serve the same purpose. These spaces are not hypothetic cleavage planes to be illustrated as lines in the commonly conceived cross section but are actually capacious and definite compartments which can be distended by air injected under zephyr pressure. These spaces, demonstrable on the living body or the cadaver, come to contain and guide pyogenic products in disease The "fascia lata strip," which is removed for arthroplasties, with its one gliding surface is but a part of the wall of one of the compartments of the thigh Let us see this in a motion picture

When the skin and superficial fascia have been dissected off over thigh, buttock, knee, and leg, a dense, white glistening fascia is left surrounding the extremity. This fascia, called in various regions by different names, was termed by H. J Prentiss as "vaginal" or ensheathing fascia, for it does precisely that to the entire body. It encircles the thigh. If this fascia is now pricked a few inches above the knee on the outer side, and compressed air is permitted to enter under gentle pressure, the vaginal fascia is at once seen to balloon out. The thigh swells mightily. The entire surface floats up, carrying with it the rectus femoris and sartorius muscles from shortly above the patella to the level of Poupart's ligament The fascia is now visibly drumlike The lateral surface of the thigh has also markedly swelled, the iliotibial band (merely a thickening of the vaginal fascia) and the tensor fascia femoris muscle rising with the fasica as far laterally as the outer hamstring muscle group It is only on the medial aspect of the thigh over the adductor muscles that the vaginal fascia appears glued down to the underlying structures.

As the thigh is watched, the observer finds that inflation of the posterior aspect is now commencing. The buttock gradually distends until the heavy gluteus maximus muscle has floated out like an inflated bag. Extending down from beneath the gluteus maximus is seen a single broad inflated tube surrounding the hamstring group This tube diverges like a pneumatic trouser leg at the apex of the popliteal space to run down each side of this region to the insertion of the hamstrings, on the tibia and fibula.

It is apparent that there exists a large anterior or anterolateral compartment the entire length of the thigh which communicates superiorly and laterally with a large posterior compartment under the gluteus maximus. In direct communication with the latter space is a trouser leg portion extending down over the diverging hamstring muscles.

Anteriorly, this inflated vaginal fascia is now incised from the level of the anterior superior spine to the knee so that the walls of this compartment may be examined. Traversing these compartments are frail bridging septa of fine areolar tissue In some regions they are more numerous than in others. The picture obtained by air inflation is confirmed (Fig. 6). The muscles which floated up (as the rectus femoris and the sartorius) are in the substance of the vaginal fascia, the fascia splitting to enclose them. The rectus femoris leaves the vaginal fascia a few inches above the knee to join the vasti in the common tendon of insertion of the quadriceps Examination of the floors of the compartment shows that the vastus internus, medialis, and lateralis muscles, for example, are covered with a fine glistening layer of fascia which may be termed muscle fascia. This muscle fascia forms the floor of the compartments and is limited laterally by the insertion of the vaginal fascia on the linea aspera. When one examines the adductor muscles, however, one finds that along the medial edge of Hunter's canal the vaginal fascia is fused with the muscle fascia of the adductor muscles. In the floor of the anterior compartment is the vascular sheath and the anterior femoral nerve

Posteriorly (Fig. 7), on dividing the gluteus maximus transversely, one notes how its muscle belly, which lies between two layers of the same layer of vaginal fascia, is continued laterally from the fascia forming the roof of the anterior compartment. The anterior half of the gluteus maximus fibers insert simply into the vaginal fascia, and the posterior half of its fibers leave the vaginal fascia to insert on the gluteal tuberosity of the femur On following the vaginal fascia down, dividing it longitudinally over the hamstrings, one sees the diverging legs, each with its tube of vaginal fascia, and finds that the popliteal space is left intact. On puncturing the fascia bridging

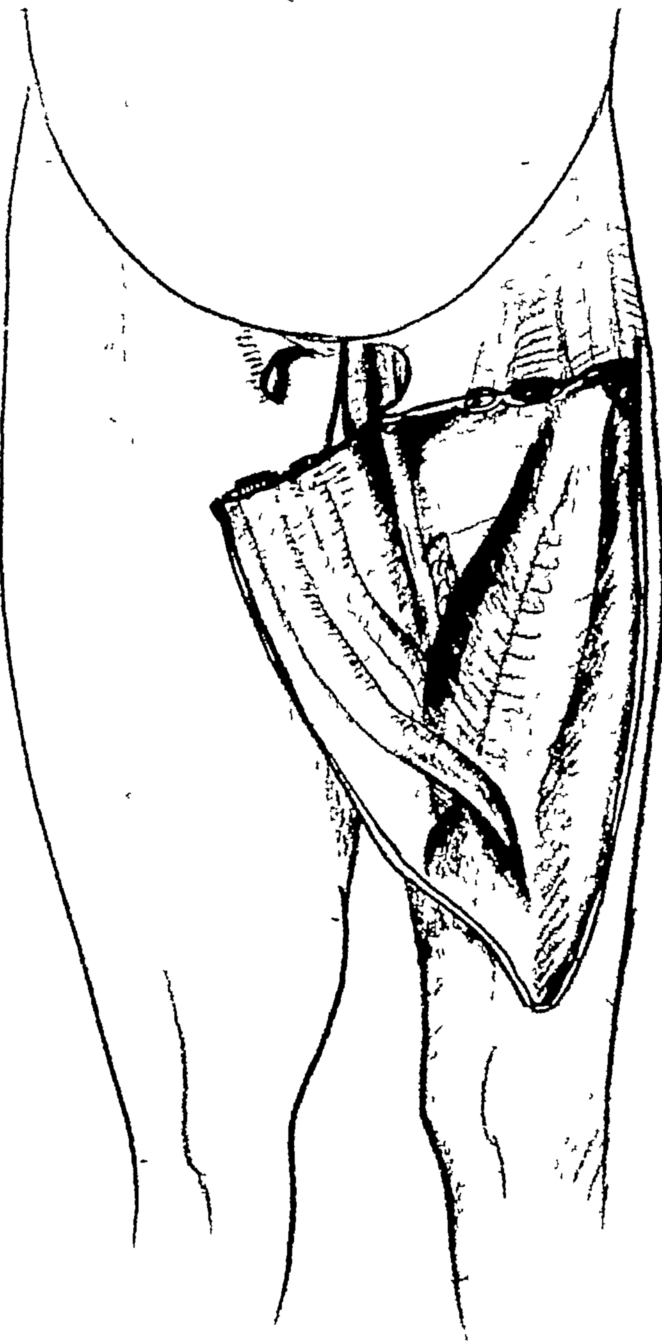


Fig 6 Antero-lateral compartment of the thigh (H. J Prentiss) Vaginal fascia has been cut and reflected with rectus femoris and sartorius muscles visible in the reflected roof of the compartment

the popliteal space and inserting the air nozzle, one finds that this space inflates together with a space on the back of the leg but that is not connected with the thigh spaces.

It may be noted especially that the anterior and posterior compartments communicate

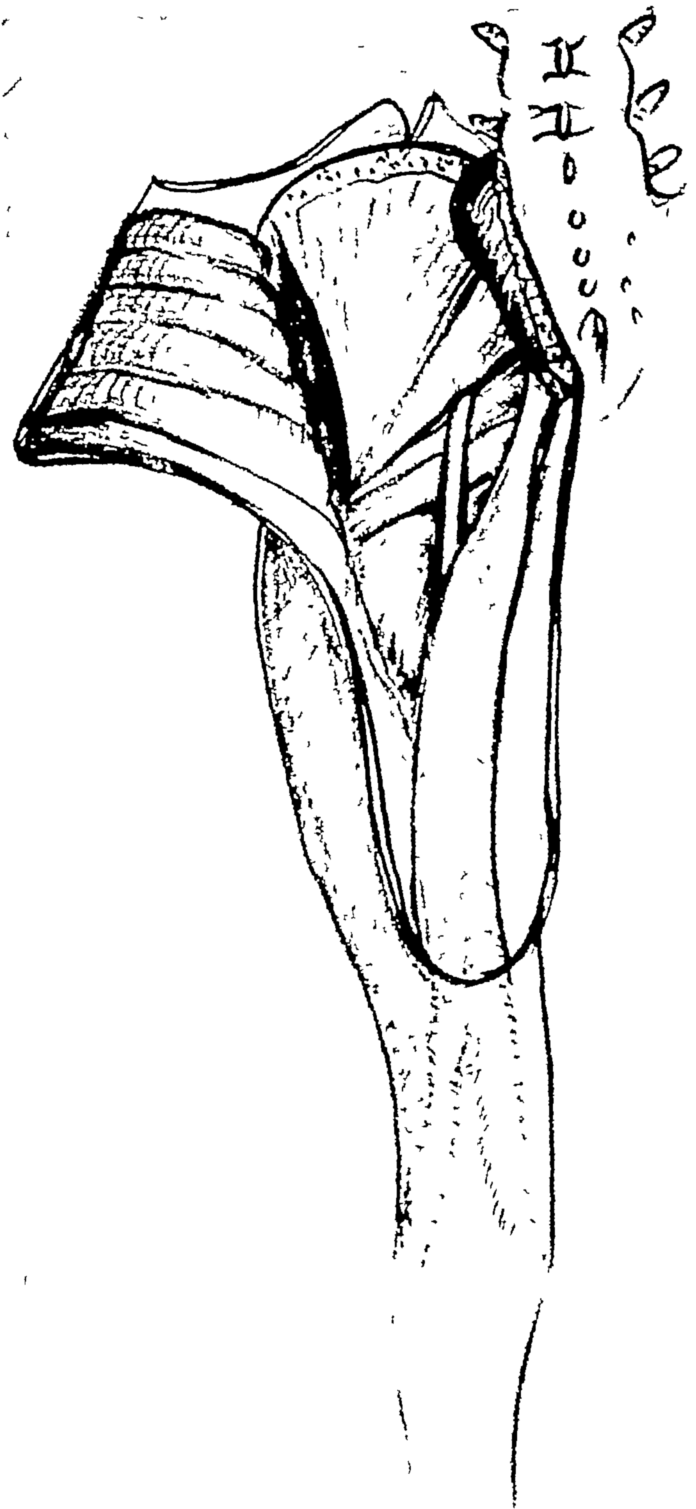


Fig 7 The posterior compartment of the thigh (H J Prentiss) Vaginal fascia of posterior aspect of thigh split and also gluteus maximus, which is incorporated in the vaginal fascia Muscle fascia of hamstrings intact Sciatic nerve visible in floor of compartment Dark space beneath reflected gluteus maximus is the communication with the anterior compartment

above and around the greater trochanter and the deep trochanteric bursa. At this point of transition, the roof is the vaginal fascia containing the entire tensor fasciae femoris muscle and the entire gluteus maximus muscle save for one half of the fibers of insertion of the latter. The floor is the muscle fascia over the gluteus medius and minimus muscles.

The salient and anatomic features of the situation may be listed thus

1) There are two large compartments in the thigh - an anterior and a posterior. They communicate over the greater trochanter under the vaginal fascia.

2) The important structures immediately beneath the floor of the anterior compartment are the anterior femoral nerve and the great artery and vein.

3) The structure of most interest in the floor of the posterior compartment is the great sciatic nerve (Figs. 8, 10).

4) Several bursae have easy access to the compartment, in particular the large, deep trochanteric bursa, which lies between the va-

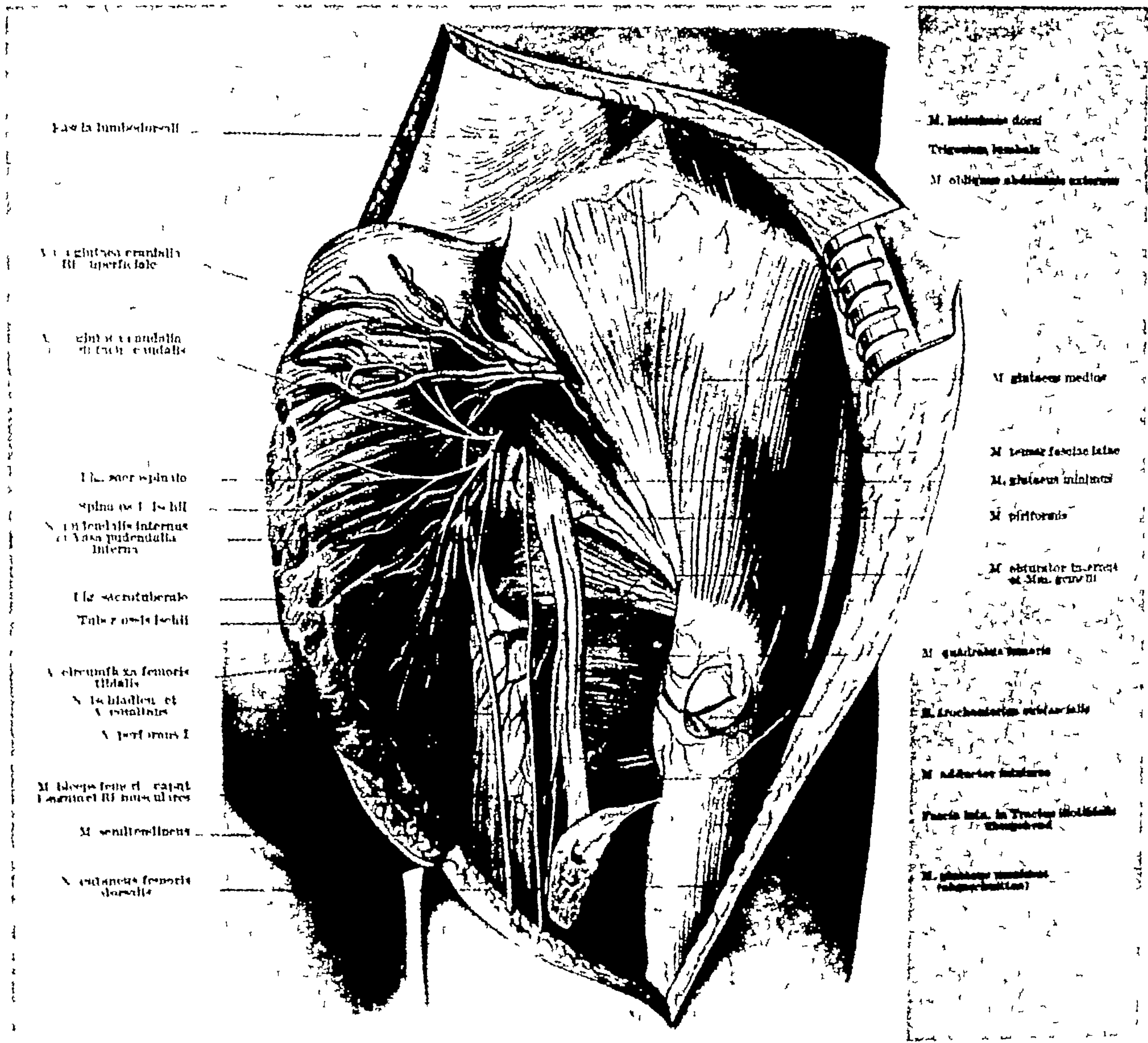


Fig 8a Structures beneath gluteus maximus Floor of posterior compartment (from V. Lanz-Wachsmith).

ginal fascia above and the greater trochanter below

5) The popliteal space belongs in a special sense to the leg and not to the thigh.

These large compartments are obviously "dead spaces" into which adjacent pathologic processes accompanied by suppuration can discharge purulent material Once in these spaces, such pus may travel a very long way into communicating spaces from psoas abscesses or iliac or femoral osteomyelitis. Hematogenous infections, so-called diabetic "cellulitis," and infected postoperative hematomas may at times involve them. Infection may be introduced with medication (as with bismuth) in the buttock, or with shrapnel fragments, or with hypodermoclysis where a fatal B. Welchi infection was observed The compartment

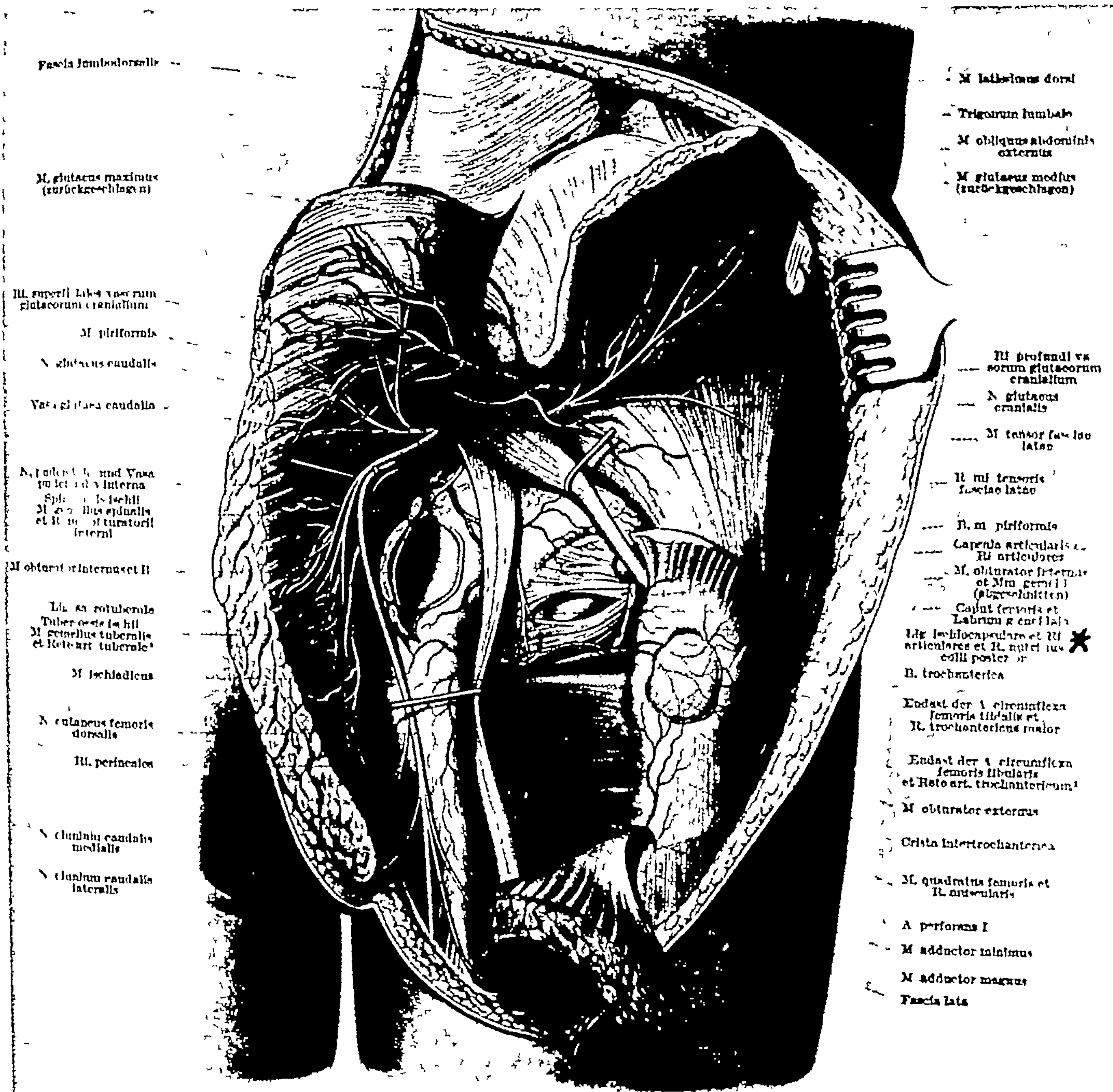


Fig 8b Deeper layers beneath floor Posterior articular nerve supply marked (*) (from V Lanz-Wachsmith)

walls may be reduced to thick avascular rigid scar covered with granulation tissue, and their contents may be secondarily involved - particularly veins and nerves. Bed sores, buttock and trochanteric, frequently extend into the compartments by erosion terminally In each case surgical recognition of the lesion will lead to efficient drainage of both the primary suppurative lesion and the compartments involved (Fig. 9).



Fig 9 Space collection persisting after the acetabular lesion (focus) has healed, outlined through sinus

Incisions

Long, lateral, linear incisions adequately open the anterolateral space. To incise the posterior *compartment under the gluteus* maximus one curves the upper end of the fascial incision backwards above the greater trochanter. Then one cuts free all the gluteus maximus fibres inserting into the ileotibial band and the upper two-thirds *of the muscle* fibres inserting on the gluteal ridge of the femur.

This principle of opening the fascial compartments and folding back hinged muscles, as the gluteus maximus, is a logical method of obtaining adequate surgical thigh exposure for purposes other than evacuating infections without sacrificing valuable structures

For example, to expose the buttock, one may split the vaginal fascia or fascia lata along the upper edge of the gluteus maximus, freeing it. Now divide the fascial thickening longitudinally, freeing the fascial insertion of the gluteus maximus. If one now curves backward and distally and incises down on a clamp inserted between the remaining fibres of the gluteus maximus and the femur beneath, the large muscle will be comparatively free, so that it may be cautiously hinged back on its neuro-vascular pedicles, thus exposing the floor of the posterior compartment in the buttock The sciatic nerve and the small external rotators of the hip, etc., will be available should one be studying posterior sensory nerves innervating the hip joint (Fig. 8b) If the incision be carried distally in the mid-line of the posterior thigh, one may displace the bundle of the hamstrings to follow the sciatic nerve distally. The small sciatic nerve which lies on the muscle fascia over the hamstrings in the mid-line of the thigh should be seen and left uninjured

The incision as above outlined is essentially the question-mark incision of B. Stookey, and details of its performance should be studied by all thigh surgeons in the extraordinarily lucid and well-illustrated text of A. K. Henry In this text the reader will find the principles of splitting the fascia longitudinally and retracting the mobilized superficial layer of muscles applied to exposure of the femoral shaft from the front Remember it is the anterolateral space that is opened when the "seam *has been* ripped" and the mobile rectus has been retracted in its fascia, thus exposing the "silvery" investing muscle fascia of the vastus intermedius in the floor of the space. By longitudinally splitting the intermedius off *from the vastus lateralis, the shaft* is exposed up to the lateral circumflex vessels and the nerve to the vastus lateralis. If the perforating vessels are located by retracting the vastus lateralis, bleeding can be minimized.

Hip joint approaches will be described by others. We may remember that by splitting the fascia lata downward from the anterior-superior iliac spine in the interval between the

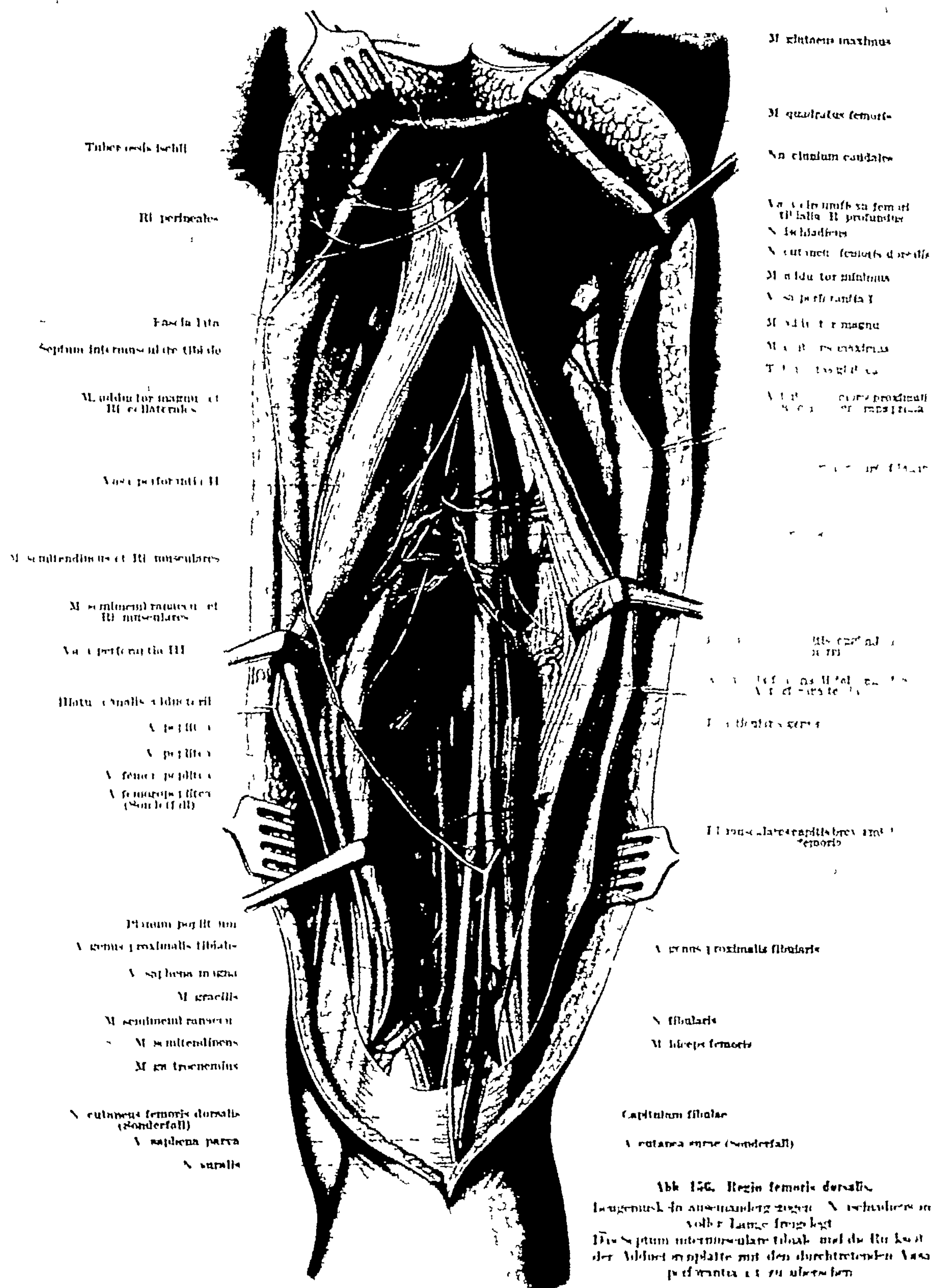


Fig 10 Structures of posterior thigh (from V Lanz-Wachsmith).

tensor fascia femoris and the sartorius, we are again opening the anterior compartment. We can therefore prolong a Smith-Petersen incision distally to expose the femur below the hip level.

To perform surgery on the femoral shaft, most men prefer lateral and anterolateral approaches Medially, the thigh is a compact structure, and incisions will here usually be direct dissections of the vascular sheath, using the sartorius belly as our guide to the roof of Hunter's canal

Attention should be called to the posterior incision of D. Bosworth and the mid-line and lateral popliteal incisions of A K. Henry, which provides most valuable and direct approaches to the back of the thigh bone. Time permits only their mention here (Fig 10).

Power of Thigh Muscles

We may reflect on the comparative power of the major thigh muscles as indicated by their weights (Frohse and Frankel). The quadriceps group in a male weigh 1870 gm. while the gluteus maximus with 525 gm. is a rather poor second If we anticipate replacing a paralyzed quadriceps with transposed hamstrings, we should note that the biceps weighs 305 gm. and is the fourth heaviest muscle in the lower extremity The semimembranosus weighs 320 gm., and the semitendinosus, 200 gm. In all, the hamstrings weigh 820 gm., were it possible to spare them all, which is rarely the case. As for hoping to replace palsied hip abductors adequately with an intact tensor fascia lata by transplantation, one compares the 115 gm. tensor with the sturdy gluteus medius (325 gm.) and the gluteus minimus (186 gm.), which total 511 gm. The function of the medius in shifting balance has called forth the name of the "dancing muscle." Its task is arduous as part of the "pelvic deltoid."

Ruptures of Thigh Muscles

Such powerful muscles, as might be expected, sustain complete as well as incomplete tears on sudden abnormal, violent contraction. Rupture of one or both glutei occurred in two patients. Each was injured in the same fashion - caught between a backing truck and a loading platform. Each sustained a massive hemorrhage into the fascial spaces of the thigh. In one patient both gluteus maximi underwent almost complete necrosis and fibrous replacement as seen at subsequent operation. Yet he was able to continue work as a laborer. In fact localised loss of a gluteus maximus does not appear to be a very disabling lesion, judging from wartime experience with local gunshot palsies, if the other glutei are not affected Rupture of the adductors, especially the longus produces a painless lump. Rupture of the rectus femoris in lower or mid thigh produces an actively retractile lump and weakens the power of knee extension materially. Resuture, even late, is feasible. Avulsion of the extensor mechanism at the tibial tubercle or lower pole of the patella requires prompt resuture and adequate postoperative immobilization, since separation has been observed to recur. Myositis ossificans should be mentioned.

In conclusion, since each of us has special interests in the thigh, each may hope to contribute to our knowledge of this useful member. There is much detailed anatomy yet to be ascertained. For example, how many of us are interested in variations of motor nerve distribution to the adductor magnus through the obturator and sciatic nerves? Yet F. Hark has pointed out its significance in spastic paralysis. He observed obturator neurectomy performed for scissors gait in certain severe spastic children may be followed by a distressing and permanent total hip abduction contracture unless the distribution of voluntary residual control of adduction from the sciatic nerve is checked before the obturator nerve is sectioned. If the patient can voluntarily flex his knee, he has intact hamstring innervation (through the sciatic) and therefore very probably has intact sciatic branches supplying the adductor magnus as well. Such a patient may safely lose his obturator supply to the magnus as well as all innervation to the adductor longus and brevis. The residual sciatic supply to the magnus will suffice to prevent the occurrence of a crippling (bilateral) abduction deformity.

REFERENCES

1 Braus, H. Human Anatomy (German) Vol I Berlin, J Springer, 1929
2 Frohse, Friedrich, and Fränkel, Max Muscles of the Human Extremities (German), in K. H von Bardeleben's Handbook of Human Anatomy (Jena, Gustav Fischer, 1913) Pt. 2, v 2, p 415-693
3 Grant, J C B A Method of Anatomy Baltimore, William Wood & Co., 1937
4 Hark, F Personal communication
5 Haymaker, Webb, Woodhall, B Peripheral Nerve Injuries Philadelphia, W.B Saunders Co , 1945
6 Henry, A K Extensile Exposure Applied to Limb Surgery. Baltimore, William Wood & Co (Reprint edition 1946)
7 Milgram, J. W Surgery of Suppuration in the Fascial Spaces of the Thigh. J A M A. 98 117 - 122 (1932).
Milgram, J E Acute Subgluteal Bursitis. J. Iowa S. M. Soc. March, 1930
8 Prentiss, H J. Personal communication
9 Steinmann, F Nail Extension in Fractures (German) Stuttgart, Enke, 1912, p 44-45

Course No. 13

ROENTGEN DIAGNOSIS IN THE EXTREMITIES AND SPINE

Lecturer

Albert B. Ferguson, M.D., Roentgen Diagnosis in the Extremities and Spine

ROENTGEN DIAGNOSIS IN THE EXTREMITIES AND SPINE

(Figures at end of article)

Albert B Ferguson, M.D.

THE PROCESS of roentgen interpretation of bone and joint lesions can be reduced to a search for the presence or absence of certain specific features which are known to be useful in differential diagnosis. To become more expert in this method of roentgen diagnosis is merely to become more familiar with a greater number of specific features which are useful in diagnosis. A few of the most commonly useful of these features will be discussed as examples.

Calcification and Ossification

Calcareous material visualized in the roentgenogram may be divided into two types defined and named as follows:

Ossification--This is calcareous material which developed osseous structure directly as it was deposited No portion of the deposit is amorphous structureless material

Calcification--This is calcareous material which is amorphous and structureless at first and develops osseous texture only slowly and indirectly.

If one accepts these definitions and learns to place calcareous matter visualized in the roentgenogram in one or the other of these classes, a very useful diagnostic aid is acquired because the ossifications will be found only in growth, repair, and tumor formation while all other calcareous deposits will be calcifications.

One may, for example, see a calcareous mass projecting from the femur. If it is ossification it is a tumor, an exostosis, or ostema. If it is calcification it is not a tumor but a deposit in the soft tissues, an ossifying myositis.

Reactive and Inert Subperiosteal Calcification

Calcification beneath healthy periosteum is fusiform in shape, smooth in outline, and even in density Beneath periosteum which has been the seat of inflammation due to infection the calcification is irregular in shape, outline, and density. These irregular "reactive" calcifications are, then, evidence of present or previous infection while the smooth "inert" subperiosteal calcifications indicate merely some form of stasis or hemorrhage.

Swelling and Soft Tissue Mass

Thickening of the soft tissues may be circumscribed and sharply defined at the ends, forming a soft tissue mass, or it may be prolonged axially and poorly defined at the ends, forming a swelling. If a mass as thus defined projects from a bone it is a reliable indication of malignancy, whereas the swellings occur in trauma, stasis, and infection.

The subcutaneous tissue over a swelling may exhibit an abundance of delicate vascular markings. This characterizes reactive swelling, which indicates active infection. Acute infection in a bone or joint should, in fact, be recognized roentgenographically within forty-

eight hours by means of this reactive swelling, providing it has not been restrained by chemical treatment.

Invasive and Infiltrative Active Destruction

When destruction is progressing actively in a bone, there is an area of loss of substance the surface of which appears minutely spiculated with calcareous density fading out over an appreciable area. This is the appearance of active destruction It is seen only in malignancies and active infections. When the destruction is irregular with some bone in the involved area as yet unaffected the destruction is infiltrative and the lesion infectious. When all bone in the affected area is in some stage of disintegration the active destruction is invasive and the lesion probably malignant. Thus, the type of destruction offers another and frequently useful means of distinguishing between infection and malignancy

Classes of Lesions

Even the few features which have been described are sufficient to accomplish much in the roentgen interpretation of bone lesions They can, for example, serve to classify bone lesions in the following groups 1) calcareous degeneration, 2) active infection, 3) tumor, 4) deformity, and 5) trophic lesion.

Calcareous Degeneration

Calcareous degeneration may occur in many diseases. When it constitutes the whole of the roentgenographic picture the condition is classed as calcareous degeneration. Such lesions are characterised by the presence of a dense shadow representing a calcification with no other change from the normal. Examples are seen in osteoarthritis, arteriosclerosis, bursitis, posttraumatic calcification, and several other conditions. When one is able to classify a lesion as a calcareous degeneration it is usually a simple matter to identify the specific condition by means of the test of consistency If a feature is consistent with the size, shape, and position of a structure or tissue normally present, it is safe to assume that it involves that organ or part. Thus one reaches the conclusion that the disease is osteoarthritis, fibrositis, bursitis, etc., as the case may be.

Active Infection

The presence of reactive subperiosteal calcification, infiltrative active destruction or reactive swelling indicate infection, and the swelling or active destruction indicate that the infection is active Swelling is the important feature in determining that infection has not yet subsided and in determining the area in which infection is active at any given time. When infection is no longer active it is classed as a deformity, residual from infection.

A lesion having been identified as an infection, the most important factor in distinguishing between the common infections is the density of the calcifying reactions. In syphilis the density is usually so great that calcification is indistinguishable from the cortex while in pyogenic lesions the cortex can still be identified readily through the calcification. In tuberculosis there is no calcification or calcification of only very light density. Density of calcification in infectious lesions should therefore be added to the list of useful features which have been described.

Tumor

An essential characteristic of tumors is the absence of the evidences of infection. They do not tend to develop reactive subperiosteal calcification, reactive swelling, or infiltrative destruction

Tumors may be identified as such and the benign tumors may be separated from the malignant.

Malignancy is indicated by bizarre tumor bone formation (ossification), invasive destruction, or active destruction without evidences of infection. It is suggested by soft tissue mass.

Benign bone-forming tumor is indicated by adventitious ossification. When an adventitious calcareous formation has been recognized as ossification containing normal bone elements the lesion is a benign tumor.

Benign tumors of the cyst-like type are indicated by a defect in bone without the evidences of malignancy or infection. In most cases the lesions are also characterized by expansion, that is, the cortex appears pushed out and thinned over the lesion.

Deformity and Trophic Disease

The evidences of active infection, tumor, and degenerative disease are absent in the de-

formities and trophic conditions The deformities may be developmental, traumatic, or residual from other conditions and are readily recognizable directly or by comparison with the corresponding part of the opposite side of the body. The lesions which appear to be more than a mere deformity are the trophic diseases.

Further Analysis of Lesions

We have seen that a very few features, by their presence or absence, have enabled us to classify the lesions involving bone This result developed because the features considered were selected with that end in view. The same method is to be applied to differentiating the lesions within a specific class. It is a matter of developing acquaintance, one by one, with more and more features that are reliable for distinguishing one lesion from another within the class An example has been discussed - the density of reactive subperiosteal calcification was used to differentiate tuberculous, syphilitic, and pyogenic infections. Some further examples will be offered They will be selected as indicating types of features to be considered rather than as a complete analysis of any one class of lesions.

Infectious Arthritis

Acute pyogenic arthritis exhibits the reactive swelling and syphilitic arthritis the dense calcifying reaction which have been described for the identification of infectious bone lesions. In early tuberculous and in gonococcic arthritis other features are diagnostic. The features are effusion, local decalcification, soft tissue atrophy, and thinning of articular cartilage. When all these features are present, one of these two diseases is indicated and the differentiation between them hinges upon the application of a principle which is equally helpful in many other problems

A specific combination of features may be developed to a certain degree by one disease in a certain length of time and by another disease in a different length of time. Tuberculous and gonococcic arthritis offer an excellent example Gonococcic arthritis develops decalcification, soft tissue atrophy, and thinning of cartilage to a given degree in the small fraction of the time that is required by tuberculosis The differentiation between them is therefore a matter of determining the known duration of the lesion and judging whether the features have developed rapidly as in gonococcic arthritis or slowly as in tuberculous arthritis.

Noninfectious Arthritis

A form of destruction which is peculiar to the arthritides are punched-out areas of atrophic loss of substance. The destruction is a small rounded area of loss of substance occurring at the articular margin. This is most commonly seen in rheumatoid arthritis, but is more constant in cases of gout In these two conditions the relation of changes in the soft tissues to the area of loss of substance varies In gout, the soft tissue change is a circumscribed soft tissue mass, centered upon the area of loss of substance. In rheumatoid arthritis, there is swelling and effusion, which is centered on the joint rather than on the area of loss of substance At the interphalangeal joints of the fingers these soft tissue changes are particularly characteristic in active rheumatoid arthritis The effusion produces a fusiform soft tissue thickening centered on these joints which is characteristic for the condition

Further differences between gout and rheumatoid arthritis are that in gout the bones are ordinarily well calcified, while in rheumatoid arthritis there is systemic decalcification increasing toward the ends of the extremities.

Osteoarthritis differs from these conditions in that there is no atrophic loss of substance, no systemic decalcification, and no effusion or swelling, except in so far as mechanical derangements of the joints result secondarily in traumatic effusion. The bones are usually well calcified. There is degenerative calcification forming lips and spurs at the articular margins. The joints most subject to strain are apt to be the most affected. The terminal joints of the fingers rather than the proximal joints are characteristically affected. Arterial sclerosis usually accompanies this condition since calcareous degeneration of fibrous tissue is the fundamental fault.

Malignant Tumors

The differentiation of one malignancy from another introduces the consideration of a number of types of features which have not yet been considered Examples follow

Age and sex are important diagnostic aids. In the first decade Ewing's sarcoma is the most common primary bone malignancy, while

in the second and third decades osteogenic sarcoma and Ewing's sarcoma are common. In the later decades multiple myeloma and metastatic tumors become the most common. After fifty years of age, carcinoma of the breast is the common cause of bone metastases in the female, carcinoma of the prostate in the male

Destruction in malignancy is invasive active destruction but variations within this type have diagnostic implications. For example, resorption of affected bone structure is very complete throughout the involved area in typical metastatic hypernephroma while, at the opposite extreme, involvement of bone may become very extensive before there is very much resorption of the affected bone in any part of the lesion in Ewing's sarcoma.

The site of the tumor may be an important differential point. Both the bone involved and the part of the bone involved require consideration

Ewing's sarcoma and osteogenic sarcoma tend to involve the long bones of the lower extremities, particularly the femur Carcinoma of the breast spreads principally to the vertebrae and ribs in the dorsal area. Metastasis from carcinoma of the prostate is most common in the pelvis and lumbar spine. Multiple myeloma affects frequently the vertebrae and skull but particularly the ribs.

Metastatic carcinoma in long bones usually involves the upper portion of the femur or humerus. The metaphyseal portions of long bones are principally affected by osteogenic sarcoma, while Ewing's sarcoma is usually situated nearer the midshaft. The site of epiphyseal cartilage may be crossed from the shaft side by osteogenic sarcoma but seldom to a visible degree by any other malignancy.

Summary

Numerous examples have been given illustrating the use of specific roentgen features to classify a lesion or to differentiate lesions within a class. Efficient roentgen diagnosis is a matter of mastery of the few features that are useful for this purpose rather than the attempt to remember as a whole the various pictures that may be presented by the various diseases or by a single disease in its different stages or numerous manifestations.

The Spine

The means of diagnosing lesions in the spine include the means employed for lesions of the soft tissues whenever their use is possible. It is frequently true in spine lesions that overlapping shadows and thickness of soft tissues make it impossible to determine clearly soft-tissue swelling, subperiosteal calcification, or various other changes which might be clearly evident in a lesion in the extremities.

One must rely more fully on less conclusive evidence which can be definitely evaluated. For example, the shape of the vertebral body is a very important factor in the differentiation of vertebral lesions. The wedging resulting from compression fracture is usually characteristic. There is a break in continuity of the anterior surface of the vertebral body near, but not at, its middle, and the compression and wedging involve the upper or the lower half of the vertebrae, not both. The wedging residual from adolescent wedging round back involves both the upper and lower halves of the vertebral body, and there is a depression anteriorly at the middle of the body instead of a break in outline. The wedging following destruction by tumor or infection usually involves both halves of the body and is accompanied by alteration of structure of the remnant. Wedging due to hemi-vertebrae results in a vertebral body of less than normal horizontal diameter as well as decreased vertical diameter. Narrowing of the bodies in chondrodystrophy is symmetrical throughout the body, and the shape of the body tends toward the bi-concave. Another condition in which both the upper and lower surface of the vertebral body tends to be concave with the intervertebral disc approaching a spherical shape is postmenopausal osteoporosis with spontaneous collapse of the vertebrae.

A very important feature in the differentiation of tumors from infections is the condition of the intervertebral disks. Tumors do not affect the disks but infections regularly cause their thinning.

Most infections in the spine are tuberculous. It is not always possible to distinguish other infections from tuberculosis at a single examination. Continued observation shows that pyogenic lesions run a more rapid course with earlier ankylosis than would be expected with tuberculosis. Syphilitic and typhoid lesions develop more calcareous reaction and greater

density of calcification than tuberculous lesions. Rare infections in the spine, such as brucellosis or paratyphoid infection may be impossible to differentiate from tuberculosis by the roentgen appearance alone.

Lumbosacral Strain

Mechanical weaknesses giving rise to strain occur in the spine, particularly in the lumbosacral area. These have been discussed in the Lumbosacral Symposium and will now be considered only from the standpoint of the most important features for determining lumbosacral instability. Stability at the lumbosacral joint requires that the articular facets face each other from side to side They may vary from this position through a range of nearly ninety degrees. The oblique placing of the facets is recognizable in the anterior-posterior view by the degree of overlapping of the shadows of the facets transversely. In the internal-external or stable type the overlapping of these shadows is very slight. In the anteroposterior, or most unstable type, overlapping is nearly complete Degrees of overlapping between these two extremes indicate fairly clearly the degree of obliquity With any anomaly in the lumbosacral area, such as a sacral type of development of the fifth lumbar vertebra, the stability of the facets is usually one of the most important factors in the stability of the lumbosacral junction

The lumbosacral angle may be a source of weakness and strain For this angle to be stable, the superior surface of the first sacral vertebra should not be further from the horizontal than 42 degrees in the habitual posture of the patient, and the center of gravity of the trunk, as indicated by a vertical line through the center of the shadow of the body of the third lumbar vertebra, should fall on this sacral surface or no more than one-half inch anterior thereto. The lumbosacral angle is best determined and its stability judged in the lateral view with the patient lying. On standing, the lumbosacral angle increases if the spine is normal, while the lumbosacral angle decreases if the lumbosacral area is subject to strain This is a protective reaction which may be used to determine that lumbosacral strain is actually present at the time of examination if films are made in both the lying and erect posture.

The last of the more important features to be considered at the lumbosacral junction is displacement of the fifth lumbar body relative to the sacrum. The fifth lumbar body may be prominent posteriorly in unstable spines, especially with anterior-posterior facets. It may be prominent anteriorly in spondylolisthesis. In this condition there is developmental failure of union of the laminae to the pedicles. The resulting gap between the laminae and pedicles may be seen in a lateral view or in an anterior-posterior view oblique from below upward (commonly called the 45-degree view).

Chronic Arthritis

Rheumatoid arthritis in the spine usually begins at the sacroiliac joints with irregularity of the articular surface and later calcification and ankylosis The same processes occur at the arch articulations upward from the sacroiliac joints, and calcification in the ligaments about the vertebrae eventually develops, producing the "bamboo spine." Osteoarthritis in the spine is characterized, as elsewhere, by lipping and spur formation with little or no tendency to other changes

Adolescent Wedging Round Back

One of the conditions peculiar to the spine is adolescent wedging round back, commonly called vertebral epiphysitis. This condition starts early in life It may be recognized roentgenographically as early as three years of age. The characteristic is persistence or prominence of the vascular pit and groove anteriorly at the middle of the vertebral body In affected cases this pit and groove are still visible in adolescence, and wedging of the vertebrae begins as early as seven or eight years of age but does not develop rapidly until adolescence. The apophyseal plates of the vertebrae do not begin to ossify until mid-adolescence, and irregularity of these plates, which has given rise to the term "vertebral epiphysitis," is merely a secondary and late feature of the condition The progressive wedging does not necessarily stop when growth in height is complete but may continue into the early adult years.

Scoliosis

Another condition which is peculiar to the spine is rotary lateral curvature or scoliosis. The cases may be divided into two principal types congenital, and others The reason for

this division is that in many cases of congenital scoliosis growth is symmetrical and the deformity at a later date is merely the enlarged replica of an earlier date. Other types of scoliosis usually act otherwise. The curve tends to progress at least during the period of growth in height and the deformity becomes worse. With such progressive deformity, surgical fusion for prevention of increase of curve or for holding correction may be attempted. When this is done certain problems arise which are solved principally by consideration of certain features of the roentgenogram. If fusion is to be done to hold correction or prevent deformity, it is requisite to know what portion of the curve must be fused. Consideration of cases which have been fused and observed thereafter until growth was complete has indicated the answer to this problem. When it has been determined by previous experience that fusion of certain vertebrae in a particular type of curve results in arrest of progress of deformity, and that fusion of a lesser number of vertebrae will not stop that progress, it is evident that the action of the force deforming the spine was effective in the area fused. We can say this more briefly by saying that the area fused represents the primary curve. The term primary curve as used here, therefore, means the curve on which the deforming forces are acting, or, stated differently, the curve which if fused will prevent progress of deformity. Thus we see that there is a direct connection between the treatment to be given and the determination of the primary curve as it has been defined here.

Study of cases previously fused has enabled us to state certain principles by which a primary curve may be identified even though one has not had experience with a large number of previously fused cases. If there is only one curve, it is obviously the primary curve. Usually there are two curves. With two or more curves the lower curve may be tested by the tilt test. The patient is placed in the erect posture with the pelvis on the side of the convexity of the lower curve elevated by a lift under the buttock if sitting, under the foot, if standing, which tilts the pelvis upward three or four inches. This causes the muscles controlling the spine to attempt to bring the body erect over the tilted pelvis with the head centered over the pelvis and the body in balance. If the lower curve is not primary, the muscles can do this by straightening that curve. If the lower curve is primary, it will not be possible for the muscles to straighten that curve very much in response to this test. Thus, on the tilt test we get either marked correction of the tested curve, indicating that it is not primary, or little or no correction, indicating that is is primary.

Another method which may be applied without a lot of previous experience with fused cases is the method of *reductio ad absurdam*. This method is described as follows It is an involuntary function of the muscles controlling the spine to bring the head erect, centered over the pelvis, with the body in balance, if it is possible for them to do so. In an area in which deforming forces are producing a primary curve it is not possible for the muscles to operate in this manner. They therefore operate in areas that are not affected by the primary deforming forces. As a result of this mechanism, we can state that if we assume a given curve to be primary, the spine beyond that primary curve should express the tendency of the muscles to bring the head erect, centered over the pelvis, with the body in balance, and should not go beyond the degree of curvature necessary to produce that result. We can also state that this result will be produced in the easiest possible manner, which means that most of the correction re-establishing body balance will occur low down in the spine if possible. Having assumed that a given curve is primary, if the remainder of the spine does not act in a manner to conform to these functions, our assumption is either incorrect or there is more than one primary curve. If we make this assumption with each curve in turn, we may find that one of the curves is consistent with the assumption, and that curve is the primary, or we may find that no one curve is consistent with the assumption that it is primary. In the latter case there are at least two primary curves which are usually readily recognizable by applying the same assumption to two curves in pairs. There are other ways in which primary curves may be identified but these are the most reliable for those who lack considerable experience with previously fused cases.

Having determined by one of these methods, or otherwise, that a given curve or curves are primary, we know that if surgical fusion is to be performed, all of the primary curve or

curves must be included in the fusion area. This is the minimum fusion area and it is determined before correction is attempted. It is usually advisable to extend the fusion beyond the minimum fusion area for the purpose of establishing, if possible, a fusion area with vertebral surfaces at its upper and lower ends parallel to each other and at a right angle to a line joining their centers. This is the ideal fusion area because, when this situation is obtained, there is no necessity for compensation for deformity outside of the area included in the fusion, and thus muscles relax and motion is increased even though the fusion has been extended. The ideal fusion area as described is of course decided upon after correction has been obtained. The greater the correction, the fewer vertebrae need to be added to the minimum fusion area to produce the ideal fusion area. If more than three vertebrae at each end need be added to the minimum fusion area to produce the ideal fusion area, it is usually best to stop short of the ideal fusion area in order to leave more movable joints remaining in the spine

It is essential to have some method of measuring a curve in order to determine the progress of curvature and the result of treatment. The method about to be described has proved very satisfactory over a long period of years and is quite accurate. A curve is described as extending from one neutral vertebra to another neutral vertebra with the most rotated vertebra as its apex. The neutral vertebra is one in which there is little or no rotation as shown by the relation of the pedicles to the body in the anteroposterior view, and one in which the next vertebra away from the curve is parallel to it or tilted in the direction opposite to the curve. The neutral vertebra at each end of the curve is the end vertebra. The center of the shadow of the body of each end vertebra is marked on an anteroposterior view, and the center of the most rotated vertebra at the apex of the curve is also marked. A line is drawn joining the apex with one end vertebra and a similar line from the other end vertebra to the apex, and this line is extended beyond the apex. The angle between this extended line and the first line mentioned represents the degree of curvature. This angle increases as the curve increases, and doubling the degree of curvature represents approximately doubling the severity of curvature.

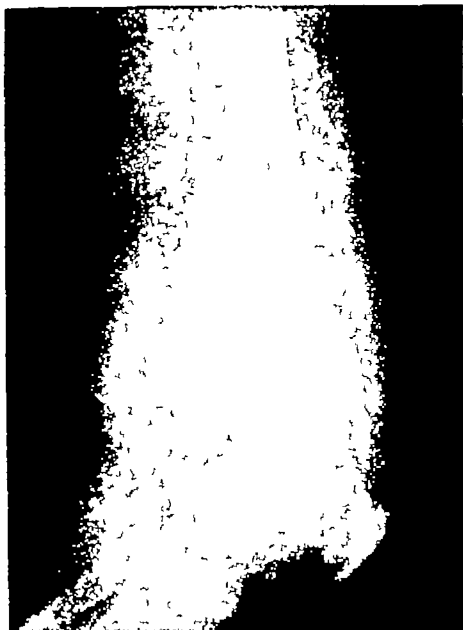


Fig. 1 Soft-tissue mass and tumor bone in a malignancy (osteogenic sarcoma)



Fig. 2. Soft-tissue swelling and reactive subperiosteal calcification in acute osteomyelitis of the fibula



Fig 3 Dense subperiosteal reactive calcification and infiltrative destruction in syphilitic lesion of the femur.



Fig 4 Infiltrative destruction, sequestration, reactive subperiosteal calcification, and swelling in chronic pyogenic osteomyelitis of the femur.



Fig 5. Invasive active destruction in the medial cuneiform bone in malignancy (metastatic hypernephroma).



Fig 6 Decalcification and ankylosis of the wrist following gonococcic arthritis The concentration of the decalcification locally at the wrist indicates that it developed rapidly, not chronically



Fig 7 Lipping of the distal joints of the fingers in osteoarthritis



Fig 8. Fusiform swelling of the proximal interphalangeal joints of the fingers in active rheumatoid arthritis



Fig 9 Thinning of articular cartilage in rheumatoid arthritis of the fingers.



Fig 10 Atrophic loss of substance of the metatarsal heads in rheumatoid arthritis This distribution of rheumatoid arthritis is usually a sequel of gonorrhea



Fig 11 Atrophic loss of substance with soft tissue masses centered on the areas of loss of substance rather than on the joints which indicates gout rather than rheumatoid arthritis



Fig. 12. Lipping of the vertebral bodies due to osteoarthritis One intervertebral disc is degenerated and thin

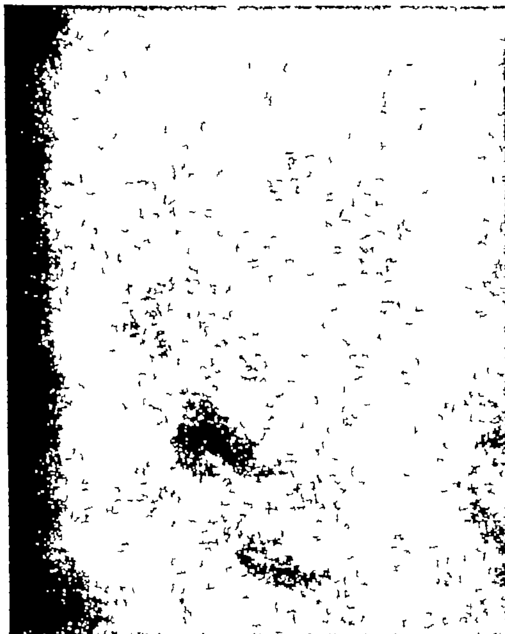


Fig 13. Adolescent wedging round back The deformity is multiple and the shape of the vertebrae differs from fracture in that both surfaces are affected and there is no depression in the superior surface nor fracture fragment at the anterior superior angle of the vertebral body

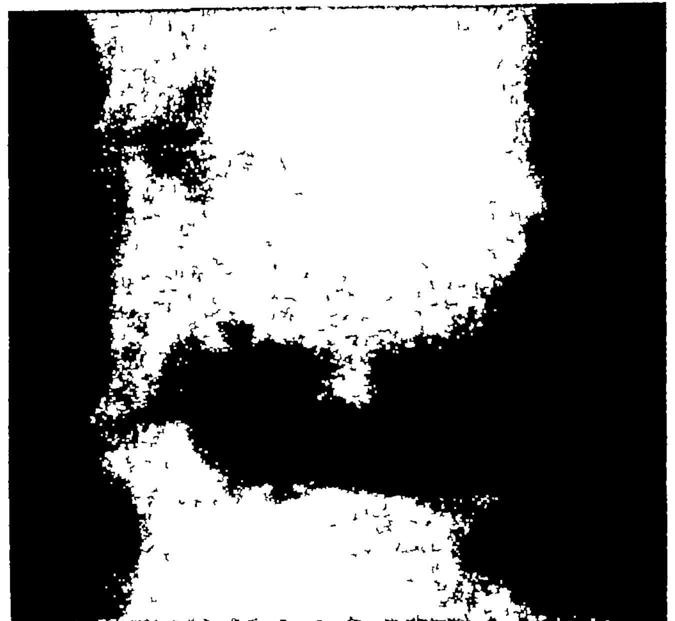


Fig 14 Accessory ossification center at the anterior superior angle of the fourth lumbar vertebra (the most common location) The ossicle cannot be imagined to fit the vertebral defect in a way which would yield a normally shaped vertebra and the shape of the body differs from compression fracture in that there is no depression in the superior surface and no fracture deformity in the anterior surface.

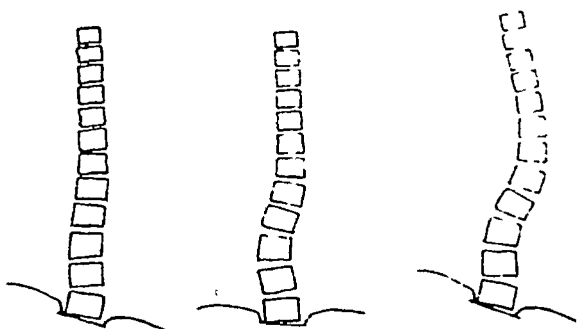


Fig 15. The tilt test The curve to be tested is diagrammed in the center Raising the pelvis on the side of the convexity of the curve causes correction of the curve in a compensatory curve as shown on your left and lack of correction in a primary curve as shown on your right

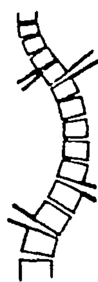


Fig 16 Measurement of a curve. The center of the shadow of the vertebral body is marked at the most unrotated vertebrae at the ends of the curve and at the most rotated vertebra at the apex of the curve Lines are drawn from the apical to the end marks and the deviation of these lines from 180 degrees is the angle of the curve.

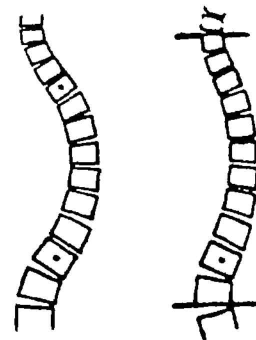


Fig 17 The ideal fusion area is obtained when fusion is extended beyond the ends of the curve to the vertebrae which have their distant surfaces parallel to each other and at right angles to the line joining their centers



Fig 18 Scoliosis before fusion



Fig 19 Same case as Fig 18, five years later, demonstrating that solid fusion in the primary area will maintain correction and, if the ideal fusion area is approached, the compensatory curves will straighten to accommodate themselves to the de-

HISTORICAL DINNER

Chairman Robert D. Schrock, M.D.

Panel

H. Winnett Orr, M.D. History of Orthopedic Surgery in the Western Part of the United States Before 1900

Mather Cleveland, M.D., and Edward M. Winant, M.D., Orthopedic Surgeons of the 19th Century in New York City

Jesse T. Nicholson, M.D., Nineteenth Century History of Orthopedics in Philadelphia

Philip Lewin, M.D., John Ridlon, 1852-1936

HISTORY OF ORTHOPEDIC SURGERY IN THE WESTERN PART OF THE UNITED STATES BEFORE 1900

H. Winnett Orr, M.D.

AN INDEX to the *Transactions of the American Orthopaedic Association* was published in 1912. Of the papers read before the Association prior to 1900, and published in the Transactions, ninety-four were by members resident in Chicago and westward to the Pacific coast.

Some of these papers by the Western men to whom we shall refer were forward looking and suggestive, because of their application, even of problems upon which we are still engaged.

For example, Doctor Edmund Andrews discussed double sacs in spinal abscesses long before the days of the x-ray. Doctor Wallace Blanchard, of Chicago, had done some of the earliest and best work in the correction of bow legs and other lower extremity deformities, by the osteoclast. Doctor F. S. Coolidge had a paper on fat embolism in orthopedic surgery.

Doctor H. P. H. Galloway, of Winnipeg, was writing papers before 1900, on deformities of the pelvis in patients with lateral curvature of the spine, and with Doctor Sherman was an early advocate of open operation for congenital dislocation of the hip. Doctor Gillette wrote early papers on pin fixation, in ununited fracture of the neck of the femur in 1898, on injuries to the spine from an orthopedic standpoint in 1900, and on traumatic spondylitis. It should be remembered also, that Doctor Gillette's campaign for the establishment of the Minnesota State Hospital, for the care of the crippled, the first of its kind, had accomplished its purpose before 1897.

Doctor Hadra, of Chicago, published a paper on the wiring of spinous processes in Pott's disease in the early 1890's. Doctor Hodgen, of St. Louis, presented papers on a scapular brace for peripheral neuritis and on Morton's operation for the reduction of club foot. Doctor Phil Hoffmann, also of St. Louis, presented a number of interesting papers just before the year 1900. The Hoffmann operation for resection of the heads of the metatarsals in claw foot was one of the first radical surgical approaches to this problem.

Doctor James E. Moore, was asking this question in 1890 "Do orthopedic surgeons operate as frequently as they should?" Perhaps we might reply, as of the present time, that the pendulum has swung too far the other way. Doctor Moore wrote also on the anatomic treatment of fracture of the neck of the femur, just a little later than did Doctor Gillette.

Doctor John L. Porter, of Chicago, pre-

in orthopedic surgery at the Childrens Hospital. Doctor Robert Watkins refers also to his father's "feud" with Doctor Hunkin which climaxed in charges against Doctor Hunkin and a hearing before the American Orthopaedic Association. Doctor Hunkin withdrew from the Association and later left San Francisco for a more secluded field.

Doctor Robert Watkins' brother was named Harry Mitchell Sherman Watkins, and Doctor R. Watkins refers now with regret to the destruction, a few years ago, of many letters of Sherman, Ridlon, and Watkins, some of which were of great historical interest.

In our neighboring state of Iowa, Dr. J. W. Cokenower had practiced a somewhat primitive kind of strap-and-buckle orthopedic surgery for many years before Steindler arrived in Des Moines in 1910. In Nebraska we also had pioneer orthopedic work. Doctor S. D. Merces (died 1907) published a little book on spinal curvature in 1878, and Doctors Lord and Jonas, also of Omaha, were interested in bone and joint surgery before 1900. Doctor Lord was Professor of Orthopaedics in Omaha for many years, and was a generous contributor to the orthopedic literature of that day

To all these Western pioneers in orthopedic surgery the profession owes a debt of gratitude not alone for their surgical contributions but for the part they played in establishing orthopedic surgery as s specialty and for the humanity and understanding they brought to the care of the crippled and deformed.

ORTHOPEDIC SURGEONS OF THE 19th CENTURY IN NEW YORK CITY

Mather Cleveland, M.D.
and
Edward M. Winant, M.D.

Orthopedic surgery in New York City began to emerge from the welter of general medical practice approximately 100 years ago. The influence of Stromyer was felt through two of his pupils who came to the city as emigrants to practice their specialty. During the second half of the 19th century there were founded in the city two hospitals whose sole purpose was the treatment of the crippled. Native born Americans, rugged individualists, some of them from the frontier, came to New York and were drawn to the practice of this specialty. Differences of opinion were the standard order of procedure and were boldly and blatantly stated. These years are marked by scenes of open strife between the doctors who practiced orthopedic surgery. Some of these battles must have approached the verbal aspect of a tavern brawl. The junior attending who questioned his chief on the slightest particular usually ended up outside the hospital, and in one instance, left the city. Occasionally the junior succeeded in overturning his chief by a sagacious lining up of the board of managers before combat.

Brain children of these prolific, rough-and-tumble doctors appeared in profusion. The subjects they wrote upon were:

1) Talipes. (A description of all varieties of clubfoot appeared in the writings of each and every one of them.)
2) Caries of the spine.
3) Hip disease. (This was a catch-all to include suppurative arthritis, tuberculosis, Perthe's disease, and doubtless, slipping of the upper femoral epiphysis.)
4) Joint contractures and deformities from any and all causes.
5) Lateral curvature of the spine and wry neck.
6) Infantile paralysis. (This began to appear in a few of their writings toward the end of the century.)

Almost without exception, there was no mention of fractures or dislocations as being in any sense the concern of the orthopedist.

In 1887 the American Orthopaedic Association was born with fourteen of these New York orthopedists serving at the accouchement. Eight of these doctors served this organiza-

tion as president before the turn of the century, a tribute to their professional ability or political astuteness, as you wish to construe it. In this pioneer organization of the thirty-eight charter members, eighteen were New Yorkers.

During this period orthopedic surgery became recognized as a specialty of medicine, both nationally and internationally.

WILLIAM DETMOLD (1808-1894 - Fig. 1)* is said to be the first physician in New York



Fig. 1. William Detmold, 1808-1894

City to associate himself with the practice of orthopedic surgery. Born in Hanover, Germany, he studied under Stromyer, the outstanding German surgeon of that time. In the year 1838 he emigrated to the United States and engaged in practice in New York City. Four years later, in 1841, he established a public clinic for the treatment of crippled children, the first such clinic to be instituted in New York. He brought American surgeons the most approved orthopedic methods of Germany and introduced the Stromyer method of tenotomy for talipes equinus deformity. He was surgeon to Bellevue Hospital in 1841, preceding Sayre by eighteen years. At the opening of the Civil War he assisted in the organization of the U.S. Army Medical Corps. In 1862 he became Professor of Military Surgery and Hygiene in the College of Physicians and Surgeons, New York. This title he held until 1865, when it was changed to Professor of Clinical and Military Surgery. He was made Emeritus Professor in 1870. His book on treatment of club-foot and analogous subjects marked an advance in the progress of orthopedic surgery.[1]

Dr. Detmold was one of the founders and the first president of the New York County Medical Association. He died at the age of 86.

LOUIS BAUER (born 1814 - Fig. 2) in recent years has been gaining the credit he deserves for his contributions to orthopedic surgery during its early period. He was forceful, energetic, and progressive and although Det-



Fig 2 Louis Bauer, M. D , F. R. C. S.

*All photographs presented are from the files of the New York Academy of Medicine

Treatment by Suspension and the Use of Plaster of Paris Bandage,[8] *and Orthopaedic Surgery and Diseases of the Joints.*[9]

Sayre directed his amazing talents not only to the field of corrective surgery but also to the field of communicable disease. He was resident physician for the city of New York, 1860-1866. He advocated compulsory vaccination against smallpox, intelligent disposal of sewage, and sanitary inspection of tenement houses. He demonstrated that cholera, instead of being a disease caused by a mysterious miasma as was thought, was communicated by human beings, and through his policy of quarantine, he restricted its spread from vessels in the harbor to the mainland.

He was one of the founders of the New York Academy of Medicine, the New York Pathological Society, and the American Medical Association He was vice-president of the American Medical Association in 1866 and president in 1880. In his presidential address he suggested the substitution of the name "Journal" for "Transactions" and the following year the name of the publication was changed to the familiar *Journal of the American Medical Association.* He is remembered because of the Sayre halter, the plaster jacket, and the Sayre dressing for treatment of fractures of the clavicle, which he devised. He remained active to the end of his eighty years, writing on orthopedic subjects up to the time of his death. It is worth noting that this outstanding pioneer failed to be elected president of the American Orthopaedic Association, while some others of less prominence, whose names are all but forgotten, served in this capacity during his lifetime.

CHARLES FAYETTE TAYLOR (1827-1899 - Fig. 5) was a Vermonter. After graduating from the University of Vermont College of Medicine in 1856, he went to London and became a pupil of Ling, the well-known "Swedish Movement" therapist. He brought with him to New York a practical knowledge of these exercises for chronic joint disease and deformities and a skill in devising braces. His careful study of cripples led him also into the field of psychotherapy, so that he was able to help many neurotic patients who were bedridden because of conversion hysteria, rather than physical incapacity. Rest, exercises, mechanical appliances, and psychotherapy were his chief remedial resources - a fairly complete repertoire for this era.



Fig 5 C. F Taylor, 1827-1899

In 1867 the New York Orthopaedic Dispensary - now the New York Orthopaedic Dispensary and Hospital - was founded to enable him to treat the crippled child by his methods, which were considered unique, and to develop scientific orthopedic surgery in this country. Theodore Roosevelt, the father of President Theodore Roosevelt, was the chief sponsor of this hospital. Prior to this, Taylor had had under his care Mr. Roosevelt's daughter Anna, who was suffering from a hip condition. Dr. Taylor remained as Surgeon-in-Chief of this institution until his retirement in 1877.

His book *The Mechanical Treatment of Angular Curvature or Pott's Disease of the Spine,*[10] was translated into German, as was his *Mechanical Treatment of Disease of the Hip Joint.*[11] His first and largest work is entitled *The Theory and Practice of the Movement Cure,*[12] published in 1861. For his ingenious apparatus to relieve deformity and movement thera-

py, he was honored with medals and diplomas at the International Exposition held in Paris in 1867, Vienna in 1873, and Philadelphia in 1876. He was made a corresponding member of the Imperial Medical Society at Vienna on Bilroth's nomination. He developed the Taylor traction hip splint and the Taylor brace, which was popularly known as the "Spinal Assistant." He retired at the age of 50 from active practice and died at the age of 72.

ADONIRAM B. JUDSON (1837-1916 - Fig. 6) was born at Maulmain, Burma, the son of the famous Baptist missionary, Adoniram Judson. He was educated in the United States, receiving his A.M. from Brown University in 1859 and his M.D. from Jefferson Medical

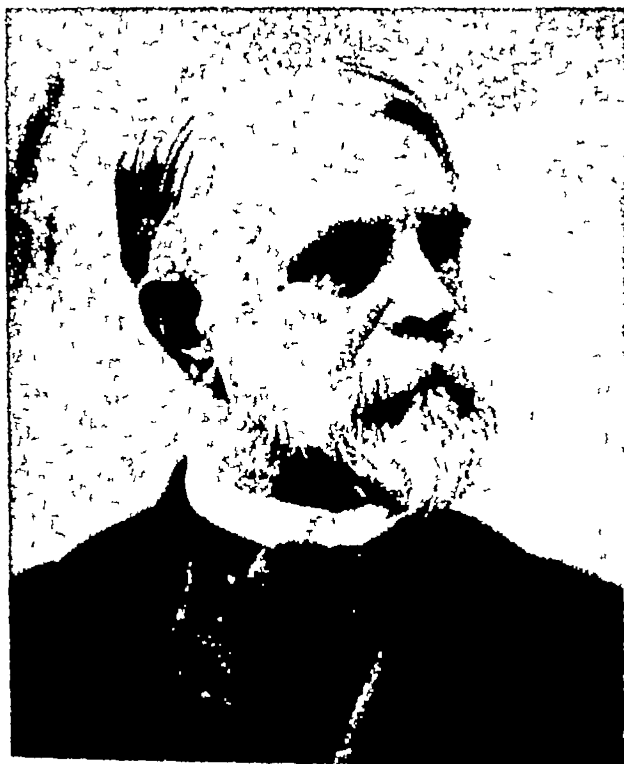


Fig. 6 Adoniram Brown Judson, M D '59, 1837-1916.

College in 1865 and from the New York College of Physicians and Surgeons in 1868. He entered the United States Navy in 1861, serving as Assistant Surgeon, and was promoted to past Assistant Surgeon in 1864, and Surgeon in 1866. On graduating from the New York College of Physicians and Surgeons in 1868, he established himself in practice in New York City and directed his attention to orthopedic surgery. He was appointed Assistant Surgeon to the New York Orthopaedic Dispensary and Hospital and Clinical Assistant to the Chair of Orthopaedic Surgery in the University of New York, Orthopaedic Surgeon to Bellevue Hospital out-patients, and Lecturer on Orthopaedic Surgery at the Women's College of the New York Infirmary.

His most outstanding contribution to orthopedic surgery was his work on lateral curvature of the spine. He demonstrated the course of lateral curvature and showed by a series of ingenious models that rotation must occur whenever the spinal column is bent laterally. His studies indicated what the principle of treatment for this condition should be. He was considered a leader in nonsurgical orthopedics. His book, *The Influence of Growth on Congenital and Acquired Deformities*,[13] published in 1905, is a very adequate orthopedic textbook for the period.

He was president of the American Orthopaedic Association in 1890 and a leading figure in the Orthopaedic Section of the New York Academy of Medicine. He died at the age of 79.

NEWTON M. SHAFFER (1846-1928 - Fig. 7) devoted half a century to the practice of orthopedic surgery. He was born at Kinderhook,



Fig 7 Newton Melman Shaffer, M D, Feb. 14, 1846

N Y, and was educated in the New York Free Academy, now the College of the City of New York, receiving his degree in 1862. During his college years he learned that Dr Knight needed a young man to undertake the duties of clerk and apprentice in the projected Hospital for Ruptured and Crippled. He applied for the position and took up his duties the day the hospital opened. Two years later he entered the New York University Medical College and graduated with honors in 1867. He was appointed Assistant Resident Physician for the Hospital for Ruptured and Crippled, served in this capacity for one year, and then opened his office. At the same time, with his brother, he started a pharmacy to help defray his expenses.

He was appointed Assistant Surgeon to the New York Orthopaedic Dispensary and Hospital in 1870, and in a few months was made Senior Assistant Surgeon with full power to conduct his work. In 1872 he was appointed Orthopaedic Surgeon to St. Luke's Hospital with a continuous daily ward service. This position created for him was the first of its kind in a general hospital in the history of orthopedic surgery.

He was appointed Surgeon-in-Chief of the New York Orthopaedic Dispensary and Hospital in 1877, a position he held for 21 years. On resigning this position in 1898, Dr Shaffer then undertook the building of a state hospital for the care of cripples. In spite of opposition, he accomplished his purpose, and in 1901 the New York State Reconstruction Home was opened in Haverstraw.

Dr. Shaffer's writings cover a wide field. His principal essays were "Pott's Disease, Its Pathology and Mechanical Treatment with Remarks on Latent Curvature of the Spine," "The Etiology and Pathology of Chronic Joint Diseases," "Brief Essays on Orthopaedic Surgery," "The Hysterical Element in Orthopaedic Surgery," all reprinted in *Selected Essays on Orthopaedic Surgery.*[14]

Dr. Shaffer is also credited with developing an apparatus by which intermittent traction or stretching could be applied in the reduction of clubfoot and other deformities of the foot. This was widely used at this time.

Shaffer founded the first New York Orthopaedic Society, now the Orthopaedic Section of the New York Academy of Medicine. He called together 13 other New York orthopedic surgeons in 1887 for a meeting in his home to initiate plans for the founding of the American Orthopaedic Association. Shaffer was instrumental in gaining recognition for orthopedic surgery as a specialty of medicine. He lived to witness orthopedic surgery become surgery in fact. This transition he originally bitterly opposed, and continued his opposition until his death at 82 years of age.

JOHN RIDLON (1852-1936 - Fig 8) Born in Vermont, he graduated from the College of Physicians and Surgeons in 1878 and served for two years as interne at St. Luke's Hospital, New York City.

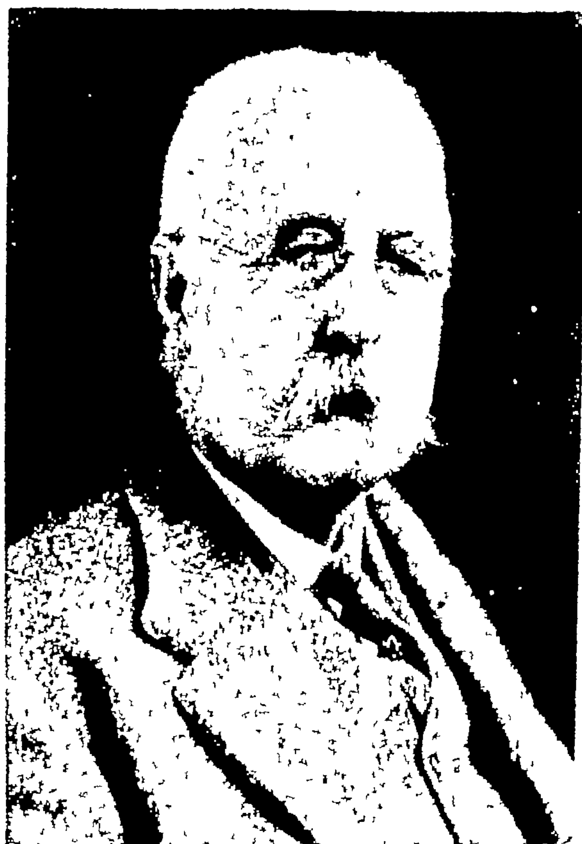


Fig 8 John Ridlon, 1852-1936

He served as Assistant Attending at the New York Orthopaedic Dispensary and Hospital. He was surgeon on the Out-Patient Staff at Bellevue Hospital and Vanderbilt Clinic. He was Instructor in Orthopaedic Surgery at the New York University Medical School.

In 1881 Ridlon was appointed Assistant to the Attending Orthopaedic Surgeon (Shaffer) at

St. Luke's Hospital. This position he held until 1887 During the latter part of this period he became interested in the work of Hugh Owen Thomas, whom he visited in Liverpool. Ridlon was one of the group of 14 New Yorkers who met in Shaffer's home to launch the American Orthopaedic Association, and he became a charter member at its first meeting in 1887.

In January 1888 Ridlon was appointed Attending Orthopaedic Surgeon to St. Luke's Hospital and served for one year only. His brief tenure culminating in his resignation was due to a bitter controversy with his chief, Newton Shaffer. Ridlon under the spell of Hugh Owen Thomas had the temerity to question Shaffer's judgment.

The minutes of the Medical Board of St. Luke's Hospital in December of 1888[15] reveal that an investigating committee reported this controversy in detail. Dr Shaffer accused Dr. Ridlon of not using the "American method" of treating hip joint disease, namely this was traction. Dr Ridlon admitted that he used it only when indicated. The committee found both doctors highly qualified and inferred that their differences might be settled among themselves. Dr. Shaffer then submitted a second complaint that Dr. Ridlon would follow no advice of his and he could not work with him. Shaffer's ultimatum served his purpose of forcing Ridlon's resignation, as he was not re-appointed, while Shaffer continued for 40 years until his death, as Consulting Orthopaedic Surgeon

Ridlon shook the dust of New York from his feet, moved to Chicago, and in 1894 the American Orthopaedic Association elected as its president - Dr John Ridlon of Chicago.

VIRGIL P. GIBNEY (1847-1927 - Fig. 9) was born in Nicholaville, Kentucky. He graduated from Bellevue Medical College in 1871 It was during his last year at this institution that he came under the influence of Lewis Sayre.

It was strange that having trained under Sayre, surgically minded for this period, that he should associate himself with Dr. Knight, who was no surgeon. He accepted an appointment as House Surgeon at the Hospital for Ruptured and Crippled upon his graduation, a position he held for 13 years. During these years he had an opportunity to observe a great deal of clinical material. He developed ideas of his own as to treatment, despite the fact that he was unable to carry out any of them. In



Fig 9 Virgil Pendelton Gibney, M D., Surgeon-in-chief, 1887-1924

1884 he published a book *Diseases of the Hip*. In this he advocated methods of treatment entirely contrary to those he had been required to practice under Knight. Consequently, his resignation from the staff of the hospital was immediately requested and obtained by his chief.

The following three years he spent in travel and study in Europe and in practice. In 1887, on the death of Dr. Knight, he was recalled as Surgeon-in-Chief to the Hospital for Ruptured and Crippled.

Dr. Gibney became a representative of the progressive group of orthopedic surgeons of this era, opposing the mechanical therapists who steadfastly denied that surgery had any place in orthopedics. He was one of the founders of the American Orthopaedic Association in 1887, and president at its first meeting. At the annual meeting of this Association in 1891, he read a paper entitled "Orthopaedic Surgery, its Definition and Scope."[17] This was an attack

on Newton Shaffer Gibney stated his ideas of the qualifications of an orthopedic surgeon, which were obviously opposed to those expressed previously by Shaffer He said "The orthopaedic surgeon should be first a well-trained surgeon and then know how to use braces, traction and plaster of paris " By modern standards, Gibney's surgical training was very meager and his surgical accomplishments left something to be desired

Through the years Dr. Gibney distinguished himself as a teacher. He was responsible for the training of many doctors who have been, and are today, leaders in this branch of surgery. He continued as Surgeon-in-Chief of the Hospital for Ruptured and Crippled until 1925. He was the only member to hold the presidency of the American Orthopaedic Association for more than one term, serving as its first president in 1887, and again in 1912. His obituary in the *Journal of Bone and Joint Surgery* ended "Gibney is dead One of the fathers of orthopaedic surgery has been called away The world is poorer by his loss "[18]

ABEL M PHELPS (1851-1902 - Fig 10), another Vermonter, was born at Alburg Springs, Vermont. He matriculated at the University of Michigan, receiving his doctor's degree in 1873 Following a period in Germany, he settled in New York City and established himself in practice He was appointed Professor of Orthopaedic Surgery at the University of Vermont, the first of a long line of peripatetic professors in our specialty at that University from New York, Washington, D C., and Boston. He also served as Professor of Orthopaedic Surgery and founder of the department at the New York Post Graduate Medical School and Hospital from 1887 until his death He established the Summer Home Hospital for Crippled Children at Englewood, N.J. He also served for many years as Surgeon to the New York Hospital As many other men of this generation, he devised an operation for the correction of clubfoot deformity [19] Phelps is also credited with adding the ischial ring to the Thomas hip splint He was president of the American Orthopaedic Association in 1894 and president of the New York State Medical Society in 1900. He died at the early age of 51.

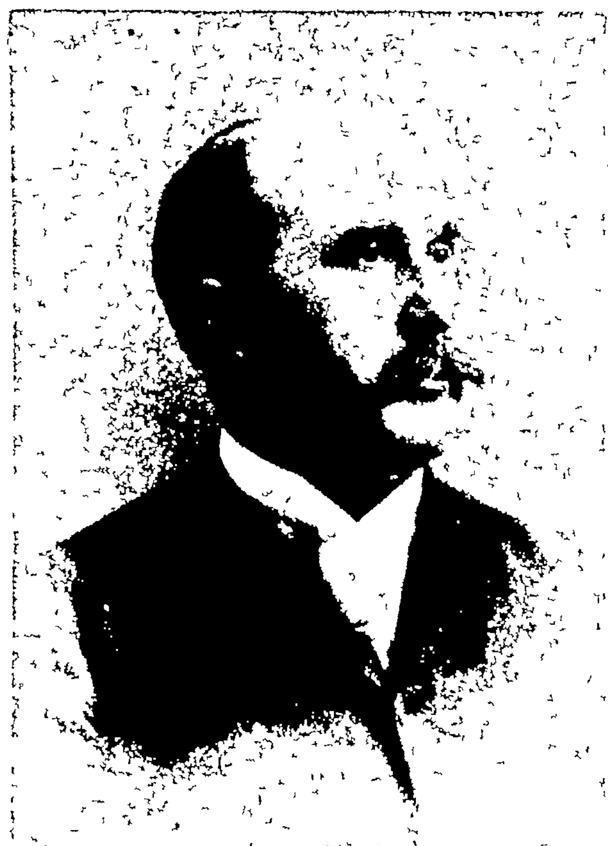


Fig 10 Abel M Phelps, M D , Jan 27, 1851

WISNER R TOWNSEND (1856-1916 - Fig. 11) was graduated from the College of Physicians and Surgeons in 1880. During 1881 he served as Surgical Resident at Bellevue Hospital. He then moved to Tennessee, where he practiced for six years In 1888 he was recalled to New York to serve as Assistant Surgeon at the Hospital for Ruptured and Crippled He became Professor of Orthopaedic Surgery at the New York Polyclinic Medical School and Hospital.

He was a prolific writer in orthopedic literature with an enormous bibliography. In 1899, at the age of 33, he served as president of the American Orthopaedic Association

Among some of his papers and articles were the following "Acute Arthritis of Infants,"[20] "Statistical Paper on Clubfoot,"[21] "Treatment of Abscesses of Pott's Disease,"[22] "The Necessity of Early Mechanical Treatment of Infantile Paralysis,"[23] "The Treatment of Hip Disease,"[24] "The Diagnosis of Chronic Joint Disease,"[25] and "Excision of the Hip."[26]

Dr. Townsend died a fairly young man at the age of 60 as the result of an accident.

SAMUEL KETCH, a New York orthopedist



Fig 11 Wisner Robinson Townsend, M D, Aug 5, 1856

of the latter part of the 19th century, of whom very little is known. The dates of his birth and death are undisclosed and no photograph has been discovered. He was Clinical Professor of Orthopaedic Surgery at the Women's Medical College of the New York Infirmary for Women and Children, Attending Surgeon to the New York Orthopaedic Dispensary and Hospital, and Attending Orthopaedic Surgeon to the Montefiore Home and Hospital for Chronic Invalids. He served as president of the American Orthopaedic Association in 1897. *Sic transit gloria mundi!*

ROYAL WHITMAN (1857-1946 - Fig. 12), a Yankee from Maine. It would be impossible to omit the name of Whitman, because of his outstanding accomplishments during the 19th century. He graduated in 1882 from Harvard Medical School. He practiced for six years in Boston. In 1889 he migrated to New York and joined the staff of the Hospital for Ruptured and Crippled.

His accomplishments were manifold and he reached the peak of his productive ability dur-

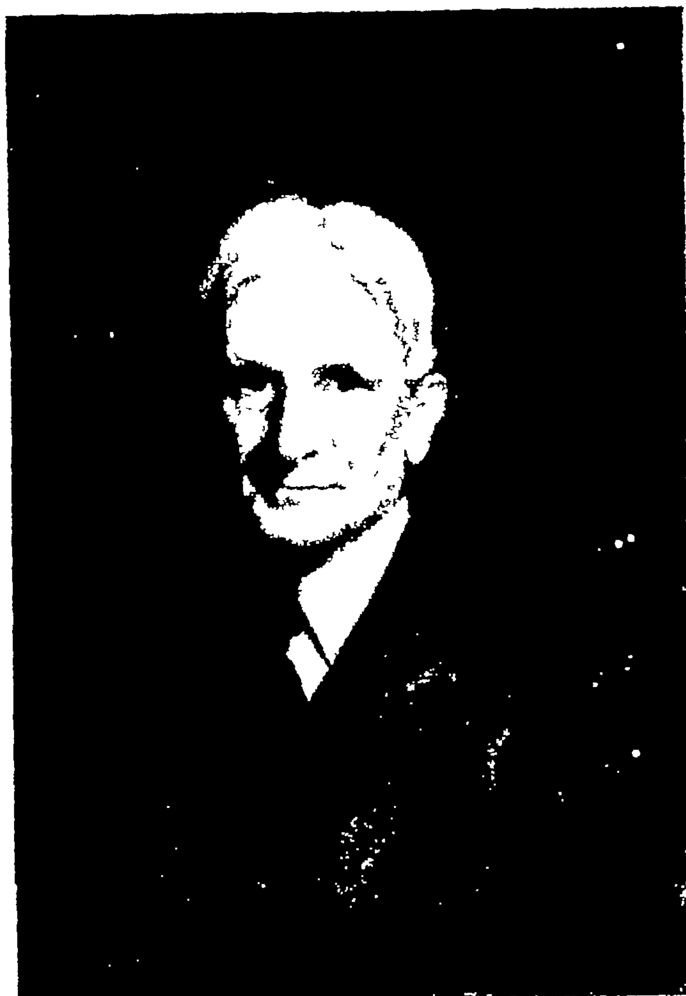


Fig 12. Royal Whitman, M D

ing the first two decades of the 20th century. He cannot be ignored as a prominent orthopedic surgeon in the most modern sense in the last decade of the 19th century. Many of his writings appeared before 1900, among which are the following *Observations on Seventy-Five Cases of Flat Foot with Particular Reference to Treatment*,[27] Additional Notes on Forcible Over-Correction in the Treatment of Rigid Flat Foot,"[28] *The Elements of the Differential Diagnosis of Pott's Disease in Childhood*,[29] and "Observations on Bending of the Neck of the Femur in Adolescents with Particular Reference to the Diagnosis and Significance of the Affection."[30]

Dr. Whitman belongs to the modern era of orthopedic surgery and is really the link between the era which we have in the main described and that in which we now practice.

Crippled New York, Privately Printed, 1939
Delavan, B D Early Days of the Presbyterian Hospital of New York New York, Published privately, 1926
Garrison, F History of Medicine Philadelphia, Saunders, 1929
Keith, A Menders of the Maimed, Oxford, 1929
Neville, R J Biographical Sketch in New York Orthopaedic Hospital and Dispensary Library
(Ridlon, J). J Bone & Joint Surg Vol. 18, No 3, (July, 1936)
(Whitman, R) J Bone & Joint Surg Vol 28, No 4.
Young, J K Manual and Atlas of Orthopedic Surgery, Philadelphia, Blakiston, 1905

NINETEENTH CENTURY HISTORY OF ORTHOPEDICS IN PHILADELPHIA

Jesse T. Nicholson, M.D.

The nineteenth century history of orthopaedics in Philadelphia originated with PHILIP SYNG PHYSICK (Fig 1). Physick was the first Professor of Surgery at the University of Pennsylvania. He held that chair for fifteen years. He was described by his biographer, Samuel D. Gross,[1] as "a cold, dyspeptic, pessimistic, unsociable man but full of sympathy for suffering humanity. Strikingly erect, and handsome, but pallid, his face was as if it were chiselled out of marble. The eyes black and his hair powdered and worn in a queue, fond of money but never claiming high fees, he yet left nothing of his large fortune to the advancement of medicine. His mind was troubled with theological matters, but what conclusions he came to in the end his reserved nature did not allow him to disclose."



Fig 1 Philip Syng Physick

Physick's father was the receiver general of the Province of Pennsylvania. Following the Revolutionary War his father became agent for the Penn Estates He intended to make his son a physician, although Physick expressed strong objections. Following his graduation from the University of Pennsylvania in the arts department in 1785, he began studying medicine with Adam Kuhn. At this time, his biographer states "he was a faithful, scrupulous, toiling soul, something of a prig and not popular with his mates, but readily devouring any mental pabulum offered him, notably when, advised to read Cullen's first lines on the 'Practice of Physic' he learnt by heart the dreary stuff."

In 1789 he was sent to London where he lived with John Hunter. He served as house surgeon at St. George's Hospital for one year. During this time he was made a member of the Royal College of Surgeons. For some reason not understood, he refused a partnership with Hunter and gave up the opportunity of working with Ashley Cooper, Abernethy, and Home He continued his study of medicine in Edinburgh and took his M.D degree there when 24 years of age.

The following year found him volunteering his help in Philadelphia for the yellow fever epidemic. In 1800 he was lecturing on surgery at the University of Pennsylvania. He was the first man in America to use absorbable suture material.[2] These were of horse hide, buckskin, and parchment. He became interested in the treatment of coxalgia and many of the well-known appliances of today are said to be modifications of his apparatus. He improved the Desault splint for fractures of the thigh He made an appliance for the outward displacement of the foot in Pott's fracture. He is credited with a rather unique method of obtaining union in an ununited fracture of the mandible. He passed a seton between the bone ends which acted as an irritating stimulus for the production of callous [3] He died in 1837 at the age of 69.

The next pioneer in orthopedic surgery was JOHN RHEA BARTON (Fig. 2), whose name



has been perpetuated by the p
surgery at the University of
which was endowed in his hono
in Lancaster, Pennsylvania, i
the nephew of Benjamin Smith
nent botanist and professor of
at the Pennsylvania Medical Sc
to the custom of the times he w
an apprenticeship at the Pennsy
for a period of five years. In
medical degree Five years lat
member of the surgical staff
vania Hospital. It is stated tha
degree of mechanical dexterity
which he directed toward the
fractures. He introduced the
which was extensively used in t
compound fractures and which p
ing place for myriads of bed b
to a biographer.[4] He devised
eight head bandage for treatme
of the jaw. He described the fra
ferior extremity of the radius,
by his name. In 1827 he publis
"On the Treatment of Anchylos
mation of Artificial Joints."[5]
a sailor who had a complete a
hip joint at a right angle. Barton
the neck of the femur to make
"The operation was done in s
and 'not one blood vessel had to
Ten years later Barton reporte
upon whom this operation was
joyed the use of his artificial
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ness (trunk-making) with great
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however, through the reverse
whose hands he had confided hi
him into a state of despondency
tion, followed by habits of intem
with all its train of evils, abuse
was, no doubt, the cause of the
afterwards took place in the art
gradually became more and m
finally, all motion ceased in t
this report he also gave an acco
condylar osteotomy to correct t

foot to the ground and my friends tell me with but a slight limp."

In 1836 a surgeon was born who described the cause for one of the most common complaints that demands the attention of the orthopedist. THOMAS GEORGE MORTON (Fig. 3) graduated from the Academic Department and the Medical School of the University of Pennsylvania in 1856.[7] He then became the

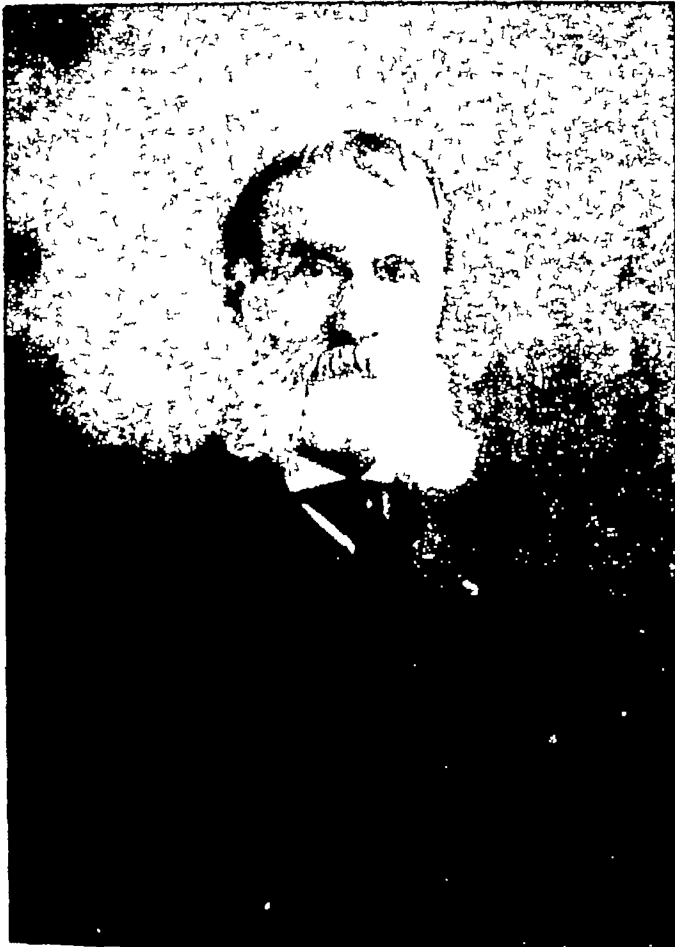


Fig 3. Thomas George Morton

resident physician at the Pennsylvania Hospital. He later was the pathologist and curator of the museum at that hospital. In 1864 he was appointed surgeon to the staff and served for over four years. He had an exceptional experience at the Mowry Hospital in Chestnut Hill, which, at the time of the Civil War, was the largest army hospital in the United States with a census of five thousand beds. During this same period he organized a second army hospital in Philadelphia. In 1867, with D. Hayes Agnew, H. Earnest Goodman, and Samuel W. Gross he founded the Philadelphia Orthopaedic Hospital. This hospital was preceded by a few years by the Boston Hospital for Orthopaedic Cases and the New York Hospital for the Ruptured and Crippled. It had a humble beginning. It was housed on the second floor of a shop of a surgical instrument maker at 15 S. 9th Street. According to the hospital records, the surgical work in the first hospital year consisted of treatment of 116 cases and 31 surgical operations. The expenses of the hospital were $1,939.54 and the receipts from the patients were $37.00.[8] The fact that the hospital survived and expanded during its first year is a tribute to the beneficence of its founder. Morton's ingenuity was expressed in several pieces of apparatus which he devised. He conceived the idea of a light truck for transferring patients in their beds from the wards to the amphitheatre. He made an apparatus for measuring inequality of leg length. He received a certificate of award by the U.S Centennial Commission in 1876 for a model ward-dressing carriage which he had invented in 1866. His surgical accomplishments were noteworthy of the period. He ligated a common carotid artery for orbital aneurism in 1864. He amputated a leg at the hip joint in 1866. He cured a case of complete osseous ankylosis of the knee by excision in 1871. He was inspired in 1886 by the loss of a brother and a son with appendicitis to do the first successful laparotomy for appendicitis with the removal of the appendix. He gave a successful blood transfusion in 1877 and he publicized the antiseptic treatment of wounds in 1886.

The affliction known as metatarsalgia,[9] or Morton's toe, he described as a clinical entity in 1875. The pain was thought to be neuralgia with radiation to one or more toes from pressure on the digital nerves as they passed between the metatarsal bones. He devised the operative resection of the metatarsal-phalangeal joint. Dr. Morton died of cholera morbus in 1903.

The name OSCAR HUNTINGTON ALLIS (Fig. 4) is generally associated with tissue forceps and a dissector. He also devised the ether inhaler. He described a sign of fracture of the neck of the femur, notably the relaxation of the fascia lata.[10] He graduated from the Jefferson Medical College in 1866 at 30 years of age. In 1896 he wrote a monograph entitled: *Inquiry into the Difficulties Encountered in*



Fig 4 Oscar Huntington Allis

the Reduction of Dislocations of the Hip He was the first clinical professor of orthopedics at the Jefferson Medical College and established the orthopedic department at that school in 1884 In 1903 he gave the Lane Lectures at the Cooper Medical College in San Francisco. Here he received an ovation for his application of the principle of the lever to articulations At the time of his death, in 1921, he had just finished a work illustrating, with models that he had devised, the functions of the spinal column with its musculature. With this device he showed the effect of posture upon normal curvature, the result of faulty posture upon the intervertebral discs, and the antagonisms of the paired muscles. The immediate outcome of this study was the investigation of the effects of desks and chairs in the secondary schools upon faulty posture

In 1887 the American Orthopaedic Association was founded. One of its founders and third president was DeFOREST WILLARD (Fig. 5). He was born at Newington, Connecticut in 1846. His studies at Yale were discontinued after two years because of eye strain.



Fig 5 DeForest Willard

Later his medical education at the University of Pennsylvania was interrupted by his volunteering during the Civil War for service with the Christian Sanitary Commission (an antecedent of the American Red Cross). After receiving his medical degree at the University of Pennsylvania in 1867 he served his surgical apprenticeship under Samuel W. Gross and D. Hayes Agnew. His training in general surgery was diverted to orthopedics by the disability of his own leg which had resulted from poliomyelitis. This physical handicap gave him an insight into the mental and physical sufferings of the crippled child that led to an unusual understanding between him and his patients. He became the first professor of orthopedic surgery at the University of Pennsylvania, and in 1889 he organized the first ward devoted exclusively to the care of the crippled children in the University Hospital. He also established the physical therapy department. He maintained a special nurse for the orthopedic department whose duty it was to visit the homes of the outpatients and supervise their care outside the hospital. This was the beginning of the Social Service Department He published some ninety orthopedic articles. He was intensely interested in the deformities that resulted from nerve lesions and wrote a number of papers

on peripheral nerve surgery. He was one of the first to approach the anterolateral surface of the thoracic vertebral bodies through a costo-transversectomy.[11] He published a book, *The Surgery of Childhood*, in 1910. Before his death, in 1910, he planned and completed the Widener School Home for the hospitalization and education of crippled children. As his biographer so aptly states, "the physical and mental rehabilitation of the crippled child was his religion and his career proved his devotion to his ideals."

HARRY AUGUSTUS WILSON (Fig. 6) was the second professor of orthopedic surgery at the Jefferson Medical College. He was elected



Fig. 6. Harry Augustus Wilson

to this honor at his alma mater in 1892 at the age of 39. In the thirteen years following his graduation he had been a clinical assistant in the medical, surgical, and ophthalmic departments at the Jefferson Hospital. He was the first to give instruction in orthopedic surgery at the Women's Medical College and the first orthopedic surgeon appointed to the Philadelphia General Hospital. He was elected Professor of Mechanical Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine.[12] He is accredited with originating the soluble compressed hypodermic tablets in 1880.[13] He devised a number of orthopedic appliances. He wrote on a wide range of orthopedic subjects in over one hundred articles, including his contributions to text books. He was president of the American Orthopaedic Association in 1901 and 1902. He died in 1919.

GWILYM G. DAVIS was born in 1857 in the Pennsylvania Dutch country. After receiving his medical degree at the University of Pennsylvania in 1879, he went to the University of Gottingen, Germany, taking his degree in 1881. During this training he was awarded membership in the Royal College of Surgeons in England. He started practice in Philadelphia as a general surgeon. His unusual knowledge of applied anatomy was evident in his book on that subject published in 1910. Earlier he had become associated with the Orthopaedic Hospital, and in 1911 he succeeded DeForest Willard as Professor of Orthopaedics at the University of Pennsylvania. His descriptions, simple English, picturing clearly the subject that he was demonstrating held his students spellbound. During the first World War he urged his assistants to enter the military service. He took on his shoulders the work that they had done. In addition he gave many intensive courses for the Reserve Corps Officers. This extra work proved too much for his rather frail physique, and in June, 1919, he died suddenly from pneumonia. He was president of the American Orthopaedic Association in 1913. He had contributed 34 articles at the meetings of the American Orthopaedic Association. Outstanding among these were "Method of Reduction of Congenital Luxation of the Hip by Manipulation," "An Operation for Ununited Intercapsular Fractures of the Hip," many papers on lateral curvature of the spine and the treatment of the paralytic foot by subastragular stabilization, which he contrived in 1907.[14] His biographer[15] says of him "A man of versatility and broad culture, he did much to raise orthopaedics to its present position among the medical specialties. To him great credit is due in bringing nearer the day when the cripple may enjoy that greatest of all blessings - mens sana in corpora sana."

REFERENCES

1 Kelly, Howard, and Burrage, Walter Dictionary of American Medical Biography, p 965
2 Physick, Philip Syng Eclectic Repertory, 6 389, (1816)
3 Physick, Philip Syng Philad J Med & Phys Sci , 5 116 (1882)
4 Kelly, Howard, and Burrage, Walter Dictionary of American Medical Biography, p 69
5 Barton, John Rhea On the Treatment of Anchylosis by the Formation of Artificial Joints N. Amer Med and Surg Jour , 3 279, 400, (1827)
6 Barton, John Rhea A New Treatment in a Case of Anchylosis Am. Jour. of Med Sci , 21 332 (Nov 1837 to Feb 1838)
7 Kelly, Howard, and Burrage, Walter Dictionary of American Medical Biography, p 877
8 Willard, DeForest P Personal communications
9 Morton, Thomas George A Peculiar and Painful Affection of the Fourth Metatarso-Phalangeal Articulation, Am. Jour Med Sci , n s 71 37 (1876)
10 Kelly, Howard, and Burrage, Walter Dictionary of American Medical Biography, p 20
11. Bick, Edgar. Source Book of Orthopedics Baltimore, Williams & Wilkins, 1937
12 Kelly, Howard, and Burrage, Walter Dictionary of American Medical Biography, p 1312
13 Bartholow, Roberts Manual of Hypodermic Medication, 1882 From Edgar Bick's Source Book of Orthopedics, Baltimore, Williams & Wilkins, 1937
14 Willard, DeForest P Subastragalar Stabilization in Paralytic Feet Am Jour of Orth. Surg 14 323 (June, 1916)
15 Rugh, J Torrance J of Bone & Joint Surg , 19 570 (1919)

JOHN RIDLON, 1852-1936

Philip Lewin, M.D

Imagine, if you will, that it is 1887 (a year before I was born). Transpose yourselves to Liverpool, England, and to a house at 11 Nelson Street

Inside this house is the office, pharmacy, and brace shop of one Hugh Owen Thomas. Thomas has 146 patients to examine sometime during the day. He must check on several back braces and foot braces, but especially hip splints. They must fit accurately, they must correct, or maintain correction. They must protect and support and minimize disability. (Thomas wrote a book on diseases of the hip, knee, and ankle.)

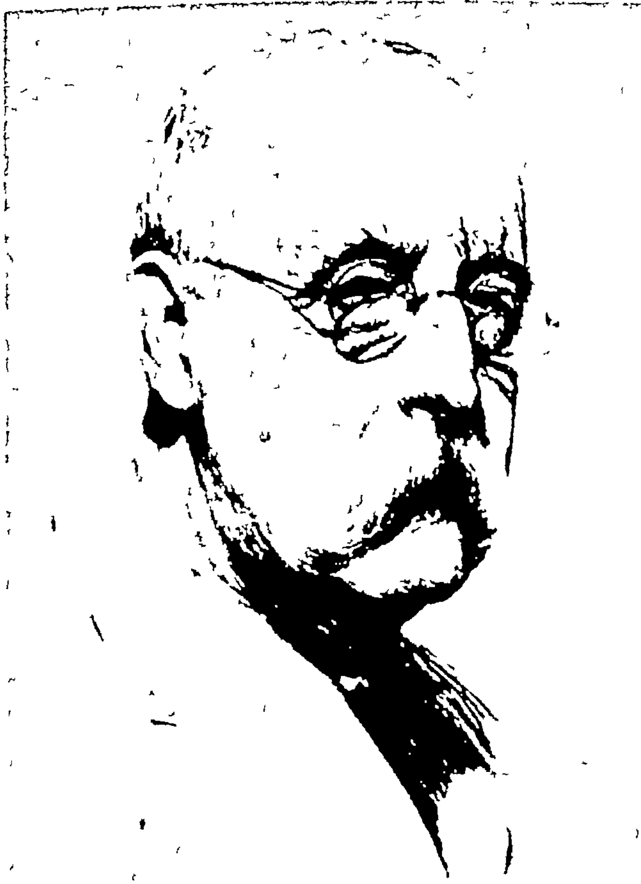
At the moment that my story begins, Thomas has called in one or two huskies from the docks to help him reduce a dislocated shoulder, without the aid of a general anesthesia, because of the danger of shock. Local anesthesia had not yet been discovered.

From a small room there emanates much noise - Thomas is correcting a club foot with his iron wrench.

A young man 35 years of age raps on the door, no answer He raps again and walks in saying, "Mr. Thomas, I am Dr. Ridlon from New York. I have come to Liverpool to find out if your results in the treatment of joint diseases are as good as you say they are in your book. I want to know whether I am a fool or you a liar."

Thomas said, "We shall find out." This was a turning point in the life of the most colorful and unforgettable character I ever knew, one John Ridlon who was born in the township of Clarendon, Rutland County, Vermont, on November 24, 1852. He was one of three boys, but the other two died in infancy. His father, Noel Potter Ridlon, was a farmer who became a dairyman just before the Civil War His mother was Nancy Bromley Hulett Ridlon, of Pawlet. Dr. Ridlon always spoke highly of his Grandmother Ridlon whose husband died at the early age of fifty-five, and who with forty acres of land and about eight hundred dollars in money at interest, raised eight children and five grandchildren.

When John was fourteen his father was killed in a railway accident. The next year he and his mother left the farm for Poultney, Vermont, where he attended a commercial college Not long afterward they went to Marengo, Illinois, making the trip over the New York Central and Michigan Central Railroads





before the days of sleepers, diners, or even wash basins on the train. At this time there came an opportunity to sell the Vermont farm, which was supposed to contain marble, and John and his mother were left in the comfortable possession of about fifteen thousand dollars.

During the next few years Ridlon worked at various times as book salesman, grocery clerk, and helper to a civil engineering corps. His mother remarried, John returned to Vermont to attend the Vermont Central Institute and later Tufts College. Through a miscarriage of college justice, he was expelled from Tufts College at the end of his sophomore year. He went to the University of Chicago, graduating in 1875.

At the various colleges he attended, John played baseball, taught boxing and fencing, organized a debating club, started a library, and pulled No. 5 on the crew. He also played bass viol in an orchestra. In 1899 he was recalled to Tufts to receive an Honorary M.A. degree, and again in 1926 to be awarded degree of Sc.B.

In 1875 Ridlon entered the College of Physicians and Surgeons in New York with Prof. E. G. Seguin as his preceptor. When he graduated, March 1, 1878, he was Class Marshal. He obtained a staff position at St. Luke's Hospital in New York, serving as house officer for two years. On June 4, 1879, he married Emily Caroline Robinson of Newport, Rhode Island. Mrs. Ridlon's father was a sea captain of many years' service. From him they inherited the home at No. 1 Sea View Avenue in Newport, where Ridlon spent the last years of his life.

Dr. Ridlon left St. Luke's Hospital in 1880 at the time their first child was born. For the next two years he was in general practice at 152nd Street and St. Nicholas Avenue. In 1880 he received his first orthopedic appointment as assistant to Dr. Newton M. Shaffer at St. Luke's Hospital, going later to the New York Orthopedic Hospital and Dispensary.

Dr. Ridlon was one of the first in America to be attracted to the work of Hugh Owen Thomas in Liverpool. He went to see Thomas' work firsthand and became one of his very few close friends and a life-long friend and associate of Thomas' nephew and successor, Sir Robert Jones. When Dr. Ridlon visited Mr. Thomas the first time, he expressed skepticism as to the results claimed for the Thomas traction splints in hip, knee, and ankle disease. Mr. Thomas was able to convince him of their utility and efficiency, however, and Dr. Ridlon became and remained the leading

American exponent of Thomas' principles of treatment for disease and injuries of the extremities. Thomas' splints and the principles he taught received world-wide recognition during the World War and led to the saving of thousands of lives and limbs.

When Dr. Ridlon returned from Liverpool in 1887 he made and applied at St Luke's Hospital the first Thomas splint ever to be used in this country for tubercular hip disease Dr. Shaffer ordered the splint removed but Dr. Ridlon refused to remove it on the ground that he was responsible for the welfare of the patient At the end of his year's service Dr. Shaffer prevented his reappointment and Dr Ridlon went with Dr. McBurney to the Vanderbilt Clinic A little later, when Dr Givney was elected to a professorship which Dr. Ridlon had expected, he moved to Chicago in 1889.

In June, 1890, Dr Ridlon returned to Europe to assist in the organization of an orthopedic section of the International Medical Congress. He called upon Grattan (of osteoclast fame) at Cork, Thomas and Jones at Liverpool, George Arthur Wright at Manchester, Florian Beely of Berlin, and many others Dr. Ridlon and T. L. Stedman of New York had been commissioned by the New York Medical Record to report the Congress. Dr. Ridlon gathered the news, while Dr. Stedman from his room at the Kaiserhof Hotel, cabled fifteen thousand words to New York, thus "scooping" all other American medical journals by two weeks!

When Dr. Ridlon came to Chicago in 1889 he was made instructor in orthopedic surgery at Northwestern University by Dr. N. S. Davis I. A year later he was made professor, and served for sixteen years. Subsequently, he was professor at Rush Medical College for three years, professor and secretary of the faculty at the Women's Medical College for three years, orthopedic surgeon at St Luke's Hospital for ten years, and attending orthopedic surgeon for a time at Michael Reese Hospital. He organized the orthopedic services of the Evanston Hospital and of the Home for Destitute Crippled Children. He was chief surgeon at the latter institution for twenty years.

Dr. Ridlon was a charter member of the American Orthopaedic Association (1887). At its tenth meeting, he was president. In his presidential address, he called attention to the presence of fifty-two members from sixteen cities and spoke of the value to the association of having not only those from the larger centers with abundant opportunities, "to see" but of having also those from the smaller towns who had time "to think " It is significant that among those from the "smaller towns" at that meeting were Gillette, of St. Paul, Griffith, of Kansas City, Moore, of Minneapolis, Packard, from Denver, Sherman, from San Francisco, Steele, from St. Louis, and Weigel, from Rochester, all of whom have placed their names permanently upon the roll of honor in orthopedic surgery.

That Dr. Ridlon dominated the affairs of the American Orthopaedic Association for many years was not only charged by others but admitted by himself. Indeed he said "When I returned from the International Medical Congress [in 1890] , I rushed back to New York to be in time for the meeting...in Philadelphia. DeForest Willard was president and George F. Ryan was secretary. I proceeded to elect A. B. Judson as president and myself as secretary. At that time the secretary was also treasurer and editor of the Transactions. I served as secretary for sixteen years and one year as president. I was counted as 'Boss' of the Association because I elected every officer except Weigel during that time." Dr. Ridlon later completed the unexpired "war terms" as secretary in 1915 and in 1918

For several years Dr. Ridlon served on the editorial staff of Surgery, Gynecology and Obstetrics and did much to assist the staff in building up the journal.

At sixty-five, September 22, 1917, Dr. Ridlon reported for active military duty at Fort Benjamin Harrison. He had already joined the Medical Reserve Corps in 1909 and had attended a Plattsburg Medical Training Camp in 1916. During his military service, Dr. Ridlon was instructor and inspector in orthopedic surgery at various forts and camps, finally becoming instructor in the Army Medical School from which he was honorably discharged February 8, 1919. Later he joined the Public Health Reserve, serving at the U.S. Marine Hospital and later at the Veterans Hospital from which he was transferred to the Edward Hines, Jr., Hospital in Chicago.

One of Dr. Ridlon's outstanding characteristics was his generosity, both professionally and financially, to younger men. There was

never a time when he would not give his energy or his money to assist especially one who was disposed to devote himself to the study and practice of orthopedic surgery. Many of the men of prominence in the Middle West and even the Far West have been Dr. Ridlon's students in orthopedic surgery, a number having been undergraduates during his professorship at Northwestern University.



In 1923 it was the privilege and pleasure of this group of students and a few other friends of Dr. Ridlon to gather in Chicago and to present to him and Mrs. Ridlon the fine portrait painted by Mr. Carl Bohnen which hangs in the Archibald Church Library of Northwestern University Medical School. At the close of the presentation exercises, Dr. Ridlon remarked that he realized for the first time that he had always had what he knew he had always wanted, namely, the friendship and regard of the men whom he had looked upon somewhat too formally as students and professional colleagues.

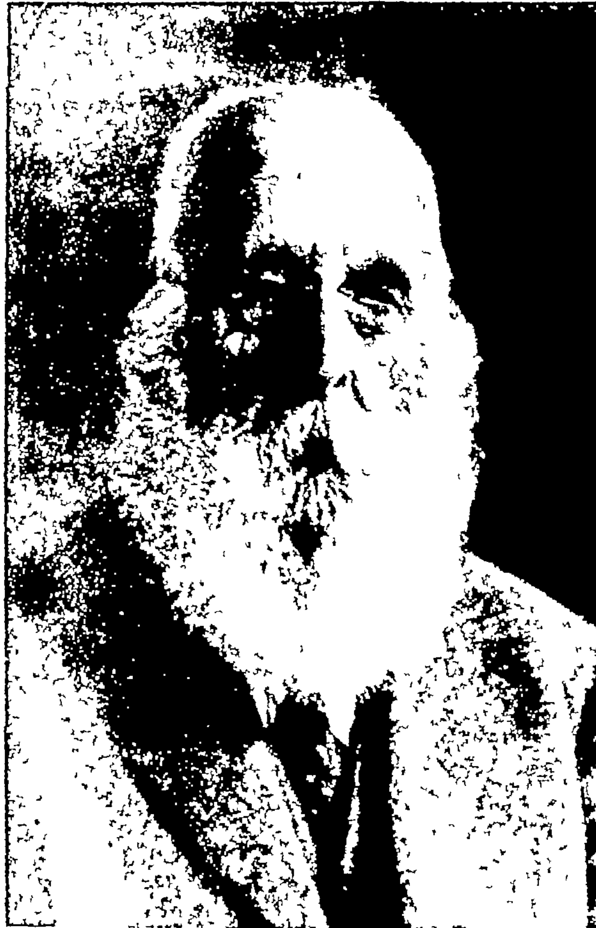
After 1928 Dr. Ridlon retired from practice in Chicago to make his home at Newport, Rhode Island, where he died on April 27, 1936 at the age of 83. However, even as he approached eighty, he remained active. During 1931 he conducted an inspection of the school children of Newport for physical defects. He traveled to meetings of his orthopedic associates in Boston, New York, and elsewhere In 1933 he was the guest of honor at the Washington, D.C., meeting of the American Orthopaedic Association under the presidency of Dr. Arthur Steindler. For several years he applied himself almost daily to typewriting notes regarding the personalities and surgical experiences of his earlier days. He kept alive an extensive correspondence with many of the men in orthopedic practice in whom he always maintained a keen personal and professional interest.



When he left Chicago, Dr. Ridlon distributed a small but choice collection of books on the history and practice of orthopedic surgery. Many of these are to be found now in the special collection accumulating under the direction of H. W. Orr in the library of the Ameri-

can College of Surgeons in Chicago The forthcoming catalogue of this collection will contain many references to Dr. Ridlon and many quotations from his letters and conversations.

Dr Ridlon's patients, pupils, and professional contemporaries always held him in high esteem 'His enemies respected him His devotion to his specialty and his ideals for the care of his patients were often expressed in criticism of those who fell short of his standards Those who profited by his teaching and by the example of his methods, however, cannot but feel that they were much the better for his industry, and for his constant inspiration



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